P246 / #634, TOPIC: *AS02 LIPIDS AND LIPOPROTEINS / AS02.13 INCRETINS AND LIPID METABOLISM.*
LIPID FRACTIONS CHANGES IN PREDICTING THE DEVELOPMENT OF NONALCOHOLIC FATTY LIVER DISEASE

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Background and Aims: Introduction. Dyslipidemia, oxidative stress (OS), and subclinical inflammation are the main mechanisms of nonalcoholic fatty liver disease (NAFLD) and hypertension (HTN) pathogenesis. Mostly lipid infiltration and lipotoxicity of hepatocytes precede the development of OS against the background of increased lipid peroxidation. Selenoprotein P (Sel P) plays a decisive role in maintaining antioxidant protection as a leading source and transporter of selenium in the body. **Aim:** to identify possible predictors and form a model for predicting the development of NAFLD in healthy individuals.
Methods: Materials and methods. The study included: main group — 49 patients (67.3% women) with NAFLD and HTN; comparison group (G2) — 51 with isolated NAFLD (58.8% women), control group (G3) — 20 individuals (55.0% women). The median age was 51.0 [45.0; 56.0] (p_{1-3} = 0.980), 52.0 [47.0; 54.0] (p_{1-2} =0.610) and 51.0 [45.0; 55.5] years (p_{2-3} = 0.564), respectively. Blood parameters were measured by standard methods. Selenoprotein P levels were measured by immunoassays (ELISA Kit). IBM SPSS 25.0 for Windows was used for statistical calculations.
Results: The studied parameters are listed in Table 1

Table 1

Levels of possibly NAFLD predictors

Index	Main group	Comparison group	Control group
Body mass index (BMI), kg/m²	27.8 [26.6; 28.5] (p_{1-2} = 0.008)	27.3 [24.2; 28.3] (p_{2-3} = 0.277)	24.3 [21.9; 26.0] (p_{1-3} = 0.049)
Systolic blood pressure, mm Hg	150.0 [145.0; 158.0] (p_{1-2} < 0.001)	125.0 [115.0; 130.0] (p_{2-3} = 0.012)	120.0 [110.0; 120.0] (p_{1-3} < 0.001)
Diastolic blood pressure, mm Hg	90.0 [85.0; 90.0] (p_{1-2} < 0.001)	80.0 [70.0; 80.0] (p_{2-3} < 0.918)	80.0 [70.0; 80.0] (p_{1-3} < 0.001)
White blood cells (WBC) × 10⁹/l	5.3× 10⁹/l [4.5; 7.7] (p_{1-2} =0.110)	6.4× 10⁹/l [5.2; 7.2] (p_{2-3} = 0.002)	5.3× 10⁹/l [4.7; 5.6] (p_{1-3} = 0.420)
Cholesterol, μ/l	**5.5 [4.8; 6.3]** (p_{1-2} < 0.001)	**5.2 [4.6; 5.9]** (p_{2-3} < 0.001)	**3.2 [2.6; 3.7]** (p_{1-3} < 0.001)
TG, μ/l	1,5 [1,2; 1,8] (p_{1-2} ≤ 0,01)	2,4 [1,7; 2,9] (p_{2-3} ≤ 0,01)	1,3 [0,9; 1,5] (p_{1-3} ≤ 0,01)
VLDL, μ/l	0,75 [0,56; 0,83] (p_{1-2} ≤ 0,01)	0,60 [0,46; 0,67] (p_{2-3} ≤ 0,01)	1,07 [0,7; 1,5] (p_{1-3} ≤ 0,01)
SelP, ng/ml	**19.7[8.0; 26.7]** (p_{1-2} < 0.001)	**43.1 [41.3; 45.4]** (p_{2-3} < 0.001)	**71.0 [54.3; 76.1]** (p_{1-3} < 0.001)

Predictors associated with the development of NAFLD in healthy individuals were determined (Table 2).

Table 2

Predictors associated with the development of NAFLD

Predictors	Enter of variables method			Backward Wald method		
	OR	95.0 % CI	p	OR	95.0 % CI	p
SBP, mm Hg.	1,066	1,009–1,127	**0,023**		—	
BMI, kg/m²	1,305	1,078–1,579	**0,006**		—	
WBC, 10⁹/л	2,303	1,279–4,148	**0,005**	7,733	1,148–52,101	**0,036**
Thrombocytes, 10⁹/L	0,724	0,606–0,866	**<0.001**		—	
Cholesterol, μ/l	7,069	2,754–18,147	< 0,001	9,944	1,433–68,990	**0,020**
TG, μ/l	15,579	3,590–67,595	< 0,001		—	
VLDL, μ/l	0,015	0,002–0,135	< 0,001		—	
SelP, ng/ml	0,233	0,127–0,429	< 0,001	0,254	0,101–0,639	**0,004**

The prognostic characteristics of the developed model are shown in table 3.

Table 3

Predictive characteristics of the developed model

Index	Value	Sensitivity, %	Specificity, %
The highest sensitivity	-3,9727	100,0	50,0
The highest specificity	1,0634	96,1	100,0
Optimal value	-0,6277	98,0	88,9
Model	NAFLD = –12,261 + + [2,045 × WBC, 10⁹/L] + + [2,297 × cholesterol, μ/l] – [1,372 × Sel P, ng/ml]		

Conclusions: The presence of reliable associations of cholesterol, WBC and Sel P allows to consider them as predictors of the development of NAFLD in healthy individuals. The proposed model has high classification characteristics and can be used as an auxiliary tool for forecasting the development of NAFLD.

P247 / #1500, TOPIC: *AS02 LIPIDS AND LIPOPROTEINS / AS02.14 OTHER.*
EFFECT OF COMBINED CARRIAGE OF POLYMORPHIC MARKERS APOE (E2/E3/E4) AND APOA1 (G-75A) ON THE APOB/APOA-I RATIO IN CAD PATIENTS IN THE UZBEK POPULATION

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Background and Aims: To compare the effect of combined carriage of ApoE (ε2/ε3/ε4) and ApoA1 (G-75A) polymorphic markers on the ApoB/ApoA-I ratio in patients with coronary artery disease in the Uzbek population.

Methods: The study included 140 patients (75 men and 65 women) with chronic coronary artery disease. The genetic typing at ApoE (ε2/ε3/ε4) and ApoA1 (G-75A) polymorphisms was performed with the PCR-RFLP method. The distribution of studied polymorphic markers in CAD patients and healthy people were in Hardy-Weinberg equilibrium.
Results: In 35 patients, carriers of the ε4 ApoE allele (group I) had a significantly higher level of ApoB (P<0.05) and the ratio of ApoB/ApoA1 (P<0.01), while among carriers of the "wild" GG genotype and carriers allele A of ApoA1 (group II), there were no differences in the level of apolipoproteins. In group I, the level of ApoB/ApoA1 was significantly higher in ε4- and A-carriers (1 subgroup, n=15) than in ε4-non-carriers (n=105) as in the case of GA/AA (n=45, P <0.01) and GG-carriers of ApoAI genotypes (n=60, P<0.05). However, in group I, when the ε4 ApoE allele was combined with the GG ApoA1 genotype (subgroup 2, n=20), the ApoB/ApoAI ratio did not differ from ε4-non-carriers due to a higher level of ApoA1 (P=0.01) than in subgroup 1.
Conclusions: Carrying the “wild” GG ApoA1 genotype can protect against an increase in the ApoB/ApoA-I ratio in patients with CAD, carriers of the ε4 allele of the ApoE gene in the Uzbek population.

P248 / #71, TOPIC: *AS02 LIPIDS AND LIPOPROTEINS / AS02.14 OTHER.*
SAFETY AND EFFICACY OF PROPROTEIN CONVERTASE SUBTILISIN/KEXIN TYPE 9 INHIBITORS AFTER ACUTE CORONARY SYNDROME; A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMIZED CONTROLLED TRIALS

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Background and Aims: Patients with a history of acute coronary syndrome (ACS) are at a higher risk of recurrent ischemia episodes. Elevated circulating cholesterol levels in patients with atherosclerotic