

abuse - (8.42 ± 3.53) years; gender distribution was: 77.9 % (n = 85) males and 22.1 % (n = 24) - females. HRS in both groups was mostly represented with the type 1: group 1 – 89.5 %; group 2 – 90.4% (p = 0.05) and had the initial scoring by CLIF-C-ACLF scale. The estimates of the probability of survival for each of the group members were found using Kaplan Meyer's procedure. That is, for group 1, the average risk of death was 0.153 ± 0.026 , and it was 0.958 ± 0.034 for group 2. Risk in group 2 increased 6.26 times compared to group 1. For the multivariate analysis, we chose those clinical and laboratory parameters which have revealed a significant correlation with the short-term mortality: age, gender, response to treatment in the first 24 hours, chronic pyelonephritis, type I of HRS and CLIF-C-ACLF score. Type 1 of HRS, response to the treatment and the high baseline score by CLIF-C-ACLF scale were identified as the predictors of the short-term mortality. Improvement in renal function during treatment was observed in most patients in group 1: a decrease of the level of serum creatinine in patients with a response ranged from 323.2 ± 91.1 to 121.6 ± 30.0 mmol/l). There were no significant differences between the two groups in terms of the treatment duration (8.2 ± 4.4 days in group 1 versus 9.1 ± 5.0 days in group 2; p = 0.05). Type 2 of HRS is more favorable for survival prognosis, as it develops more slowly and gives more time for adequate treatment measures. However, we had a very less number of such patients in our study – 10.5% of group 1 and 9.5% of group 2, as HRS type 2 is much rarer, than type 1.

The results of the study indicate that type 1 of HRS, the response to treatment in the first 24 hours, chronic pyelonephritis and high CLIF-C-ACLF score are the most important predictors of survival in patients with HRS. Monitoring of these indicators allows to identify the group of patients with the worst prognosis and to put them in priority to the liver transplantation list.

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**ALDOSTERONE SYNTHASE CYP11B2 (-344C/T) GENE POLYMORPHISM
INFLUENCE RISK OF CHRONIC KIDNEY DISEASE IN PATIENTS WITH ARTERIAL
HYPERTENSION**

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Renin-angiotensin aldosterone system (RAAS) plays a major role in blood pressure regulation. Aldosterone, synthesized in the adrenal cortex by aldosterone synthase is encoded by the cytochrome 11B2 aldosterone synthase gene (CYP11B2).

The aim of the study was to analyze the association of aldosterone synthase gene (CYP11B2) biallelic polymorphism in the promoter at position -344 (-344C/T) with Chronic Kidney Disease (CKD) in patients with essential arterial hypertension (EAH) in West-Ukrainian population. One hundred patients with EAH and target-organ damaging (2nd stage), moderate, high or very high cardiovascular risk were involved in the case-control study. Among them 79.0% (79) women and 21.0% (21) men. Their average age is 59.87 ± 8.02 ; disease duration from 6 to 25 years. Chronic Kidney Disease (CKD) was diagnosed in 29 people according to the National Kidney Foundation recommendations (2012) after glomerular filtration rate (GFR) decline <60 ml/min/1.73m² for 3 months (measured by CKD-EPI equations). All enrolled /examined patients signed the Informed Consent to participate in the research. Control group included 48 practically healthy individuals of relevant age. Gene's nucleotide polymorphism CYP11B2 (-344C/T) was examined by polymerase chain reaction.

The probability of EAH in observed people increased 1.49 times in T-allele carriers of CYP11B2 gene, but only in women [OR=1.90; 95%CI:1.02-3.54; =0.029], with contrary decreasing risk in C-allele women (p=0.041). No relevant dependences were observed in hypertensive men. Also T-allele increased probability of CKD (GFR <60 ml/min/1.73m²) in hypertensive population 1.48 times [OR=1.86; 95%CI:1.01-3.58; =0.049], especially in T-allele women 1.53 times [OR=6.51; 95%CI:1.39-30.60; =0.007] with low CKD risk in T-allele men [OR=0.15; 95%CI:0.03-0.72; =0.009], respectively. Some predictors like DM2, the 2nd and 3rd grades of Obesity, and the 3rd grade level of Blood Pressure elevation escalated the risk of CKD 2.4, 2.08-2.32 and 2.91 times as much, accordingly (p<0.05).

Thus, aldosterone synthase gene CYP11B2 (-344C/T) associates with high risk of EAH in Bukovyna region. T-allele increased risk of CKD in hypertensive population almost 1.5 times as much, especially in women.

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DEPENDENCE OF LIPID METABOLISM ON POLYMORPHIC VARIANTS OF THE GNB3 GENE IN PATIENTS WITH PRIMARY ARTERIAL HYPERTENSION

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Primary (essential) hypertension (PH) is the most common cause of left ventricular hypertrophy (LVH) and is often associated with metabolic disorders. LVH and dyslipidemia are essential risk factors and indicators of morbidity and mortality, both cardiovascular and general ones.

The aim of the study was to analyze dependence of lipid panel parameters on polymorphic variants of the guanine nucleotide binding protein (G-protein) β 3 subunit gene (GN 3) C825T (GN 3, 825C>T; dbSNP: rs5443) in patients with PH.

A cross-sectional study involved 72 patients with PH stage II, 1-3 degrees of blood pressure, high and very high cardiovascular risk. There were 29,16% (21) men, 70,84% (51) women among the patients. The average age of patients was $59,87 \pm 7,98$. The control group consisted of 48 healthy individuals of the average age ($49,13 \pm 6,28$) and sex distribution (62,5% of women, 37,5% of men). GN 3 C825T polymorphism was investigated by PRL in real time. To establish LVH, all patients had undergone echocardiography. LVH was calculated by LVMM (according to the Penn Convention) and LVMMI. To evaluate LVH, LVMMI were taken 115 g/m^2 in men, 95 g/m^2 in women (ESC, ESH 2018). The lipid panel parameters, such as: TC (Total cholesterol), G (Triglycerides), LDL-C (Low-density lipoprotein cholesterol), HDL-C (High-density lipoprotein cholesterol) were investigated in blood plasma, using diagnostic kits of the company "Accent 200" (Poland). The atherogenic index (IA) was calculated by the formula: $(\text{TC} - \text{HDL-C}) / \text{HDL-C}$.

As a result, the following lipid panel parameters in carriers of the C-allele of the GNB3 gene have been found: TC – $5,50 \pm 0,79 \text{ mmol/L}$, G – $2,10 \pm 0,8 \text{ mmol/L}$, HDL-C – $1,22 \pm 0,22 \text{ mmol/L}$, LDL-C – $4,03 \pm 0,76 \text{ mmol/L}$, IA – $3,66 \pm 0,84$. In TC-genotype carriers, patients with EH the concentration of TC was $5,82 \pm 1,15 \text{ mmol/L}$ ($p_{CC} > 0,05$), G – $1,73 \pm 0,55 \text{ mmol/L}$ ($p_{CC} > 0,05$), HDL-C – $1,30 \pm 0,21 \text{ mmol/L}$ ($p_{CC} > 0,05$); LDL-C – $4,39 \pm 1,07 \text{ mmol/L}$ ($p_{CC} > 0,05$), IA – $3,61 \pm 0,95$ ($p_{CC} > 0,05$). In C-genotype carriers, patients with EH the concentration of TC was $6,6 \pm 0,64 \text{ mmol/L}$, TG – $2,6 \pm 1,27 \text{ mmol/L}$, which was higher than in C-allele carriers according to TC – by 20,0% ($p_{CC} > 0,05$) 13,79% ($p_{TC} = 0,016$), according to TG – by 23,81% ($p_{CC} > 0,05$) 52,94% ($p_{TC} = 0,038$), respectively. The rest parameters of lipid panel have not differed significantly between genotype carriers and in homozygous carriers of the mutation T-allele had been HDL-C $1,3 \pm 0,05 \text{ mmol/L}$ ($p_{CC,TC} > 0,05$), LDL-C $4,7 \pm 0,69 \text{ mmol/L}$ ($p_{CC,TC} > 0,05$), IA $4,0 \pm 0,69$ ($p_{CC,TC} > 0,05$).

Thus, the lipid metabolism in patients with EH does not depend on polymorphic variants of the the guanine nucleotide binding protein (G-protein) β 3 subunit gene (GN 3, 825C>T; rs5443).

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EXPERIENCE OF PROBIOTICS USE IN NEWBORN WITH PERINATAL PATHOLOGY IN DYSBIOTIC INTESTINAL DISORDERS

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One of the most important mechanisms for the adaptation of newborns to the environmental conditions is the formation of non-specific protective barriers of the body, which are also physiological microbial ecosystems. The most common pathological conditions of the gastrointestinal tract in newborns are a violation of the composition and function of the colon microflora, which arise under the influence of perinatal factors and is a prerequisite for the development of inflammatory bowel diseases in the future. Alpha-1-antitrypsin (α -1-antitrypsin)