Both hypertension and obesity are key cardiovascular risk factors. Known mechanisms of combined impact of obesity and other major cardiovascular risk factors are still not clear, but the data of epidemiological and clinical studies indicate the leading role of adipokines (a group of cytokines produced by adipose tissue) in the onset and progression of cardiovascular complications in obese patients [11]. In this aspect, the recently discovered adipokines, chemerin and nesfatin-1, are of interest. Chemerin is mainly synthesized in adipocytes and preadipocytes of adipose tissue, and its expression increases in response to various factors, including inflammatory reactions and obesity. Chemerin has been shown to be involved in the development of cardiovascular diseases, including cardiovascular complications in obesity, diabetes, and metabolic syndrome [12].

Among other adipokines, nesfatin-1 has also been studied in the context of cardiovascular complications. NESFATIN-1 is another adipokine that is mainly synthesized in adipose tissue and has been shown to have anti-inflammatory and anti-atherosclerotic properties [13]. It has been suggested that nesfatin-1 may play a role in the regulation of food intake and energy expenditure, which is important for the prevention and management of obesity and related complications [14].

Recent studies have focused on the role of chemerin and nesfatin-1 in obesity-induced cardiovascular complications. These studies have shown that these adipokines can affect the cardiovascular system through various mechanisms, including modulation of vascular function, inflammation, and fibrosis [15-18]. These findings suggest that chemerin and nesfatin-1 may be new therapeutic targets for the prevention and treatment of obesity-related cardiovascular complications.

However, the precise roles of chemerin and nesfatin-1 in the pathogenesis of obesity-related cardiovascular complications remain to be fully understood. Further research is needed to clarify the mechanisms underlying the actions of these adipokines and to develop effective therapeutic strategies targeting chemerin and nesfatin-1 for the prevention and treatment of obesity-related cardiovascular complications.
perivascular and white adipose tissue, fibroblasts and platelets in the form of prochemerin. It has several active isoforms, which causes its pleiotropic effects, including prohypertensive action. A series of research has shown that chemerin has no effect on basal inflammatory status, but it promotes the production of nitric oxide and activates the PI3K-Akt-eNOS signaling pathway. The presence of endothelial dysfunction, which occurs in many cardiovascular diseases, increases the ability of chemerin to increase arterial tone, which causes the vasoconstrictor effect of this adipokytokine in the processes of vascular tone regulation and may contribute to the development of hypertension [2]. The main place of synthesis of nesfatin-1 is the hypothalamic nuclei. It is also synthesized in adipocytes, pancreatic beta cells, cells of gastric mucose and reproductive system. Nesfatin-1 is able to regulate the need for food, reduces the appetite and the amount of body fat produced, participates in the regulation of carbohydrate metabolism. Recent data suggests that nesfatin-1 is involved in the pathogenesis of hypertension, which is potentially implemented through the central system of melanocortin and oxytocin, and also expresses vasoconstrictor effects by suppressing the synthesis of nitric oxide i

Aim: To study the relationship between serum levels of chemerin and nesfatin-1 and parameters of daily blood pressure monitoring (DBPM) in hypertensive patients depending on the presence and degree of obesity.

Materials and Methods. 82 patients with hypertension, aged 60 (55; 66) years, including 26 patients with overweight, 39 with obesity and 17 patients with normal body weight, underwent DBPM. The serum levels of chemerin and nesfatin-1 were determined by the immune enzyme method using Human Chemerin and Human Nesfatin-1 ELISA kits (Kono Biotech Co., Ltd., China). Statistical processing was performed using Mann-Whitney, Pearson criteria, K-mean cluster analysis. Quantitative attributes are presented as median (Me), upper (UQ) and lower (LQ) quartiles.

Results. Serum levels of chemerin and nesfatin-1 were significantly higher in patients with hypertension (p = 0.001) compared with healthy subjects. In order to detect the joint effect of the concentration of both cytokines on DBPM parameters, a cluster analysis was performed using the K-mean method; four non-intersecting clusters were obtained with a studying error p = 0.138. The inter-cluster analysis revealed statistically significant differences between clusters in the DBPM parameters that characterize the dynamics of changes of blood pressure in the morning, namely, the rate (HRSBP and SHPDDBP) and the magnitude of the morning rise of BP (VRPSBP and VRPDBP), daytime systolic and diastolic variability of BP (VarSBP (D) and VarDBP (D)) and circadian rhythm of BP. The first cluster, where the high level of serum chemerin of 11.12 (8.2; 14.02) ng/ml was associated with high values of BMI (33.31 [30.47; 36.15] kg/m²), was characterized by the most unfavorable type of distribution of circadian rhythms of BP, VarSBP and VarDBP. In contrast, the patients of the 3rd cluster with high serum levels of both cytokines: chemerin of 7.7 (6.52; 8.44) ng/ml, nesfatin-1 of 8.96 (8.55; 9.37) ng/ml, and low BMI (25.2 (23.1; 26.8) kg/m²), had a predominant distribution of circadian blood pressure by dipper type, but high SHPSBP and SHPDDBP. The most favorable in relation to the parameters of DBPM was the 2nd cluster with moderately low content of chemerin: 4.91 (4.42; 5.26) ng/ml and high level of nesfatin-1: 8.02 (7.67; 8.43) ng/ml. A significant direct correlation has been revealed between serum chemerin and the following parameters of DBPM: SHPSBP and SHPDDBP: r = 0.35, p <0.05; VRPSBP and VRPDBP: r = 0.3, p <0.05; VarSBP and VarDBP: r = 0.34, p <0.05. There were no correlations between the parameters of DBPM and serum nesfatin-1.

Conclusions. Serum levels of chemerin and nesfatin-1 were significantly elevated in patients with hypertension. The relationship between serum chemerin and circadian rhythm, daytime variability of blood pressure and DBPM parameters that characterize the dynamics of morning changes of blood pressure was revealed. There was no convincing data on the effect of serum nesfatin-1 on DBPM indices.

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Key words: adipokines, metabolic syndrome, cardiovascular risk

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MODIFIED PROTEINS IN BLOOD OF PATIENTS WITH DRUG-INDUCED NEPHROPATHY

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Nephropathy occurs when kidney-specific detoxification and excretion do not work properly due to the damage or destruction of kidney function by exogenous or endogenous toxicants. The kidney is a major site of organ damage caused by drug toxicity. Nephrotoxicity resulting from drug exposure has been estimated to contribute to 19–25% of all cases of acute kidney injury in critically ill patients [11]. Exposure to drugs often results in toxicity in kidney which represents the major control system maintaining homeostasis of body. Understanding the toxic mechanisms...