Obesity is associated with hypertension and increased cardiovascular risk. Based on population studies risk estimates indicate that at least two-thirds of the prevalence of hypertension can be directly attributed to obesity. Although BMI as a measure of obesity is a good predictor of all-cause and cardiovascular mortality, as recently described in two separate meta-analyses [1, 2], overall mortality and especially cardiovascular mortality seems to be better predicted by abdominal or central obesity in addition to BMI.

As it’s still not possible to identify one mechanism as the dominant aetiological factor, adipokines may have a decisive influence. The physiological and pathogenetic role of adipokine apelin and its participating in glucometabolic disturbances in the article was analyzed.

Apelin is a recently discovered vasoactive peptide and adipokine that is an endogenous ligand of the APJ receptor and was named 'apelin' after APJ endogenous ligand. This G protein-coupled receptor was identified in 1993, and has a close identity with the angiotensin II type 1 receptor, but does not bind angiotensin-II. Apelin and APJ have been found to be expressed in fat tissues, heart and lungs, as well as various regions of the central nervous system. The pathophysiologic action of apelin in obesity remains unclear. It’s shown that apelin has effects not only on glucose utilization, but also apelin’s receptor is expressed in islets and apelin activation of its receptor inhibits insulin secretion. And it has been shown in clonal INS-1 β-cells that this is by activation of PI3K0phosphodiesterase 3B. Recent evidence suggests that apelin is itself expressed in pancreatic islets, particularly in β- and α-cells, raising the possibility of autocrine/paracrine effects.

There is an evidence that exogenous apelin reduces the peak plasma glucose concentration after a glucose load by increasing glucose turnover through insulin-dependent and -independent pathways. Apelin-deficient animal models have reduced insulin sensitivity and this can be corrected by the administration of exogenous apelin.

Conversely, exogenous apelin reduces the peak plasma glucose concentration following a glucose load by increasing glucose turnover and this effect is preserved in insulin-resistant animal strains. The exact cellular mechanisms leading to increased glucose uptake are incompletely understood. Apelin increases glucose uptake through phosphorylation of components of insulin-dependent pathways, such as Akt, although increased glucose uptake is still observed in the presence of inhibition of this pathway suggesting both insulin-dependent and -independent pathways.

There are some proves that chronic low-grade inflammation is thought to be key in the pathogenesis of insulin resistance, type 2 diabetes mellitus (T2 DM) and cardiovascular disease that
is associated with obesity-mediated diabetesculating levels of TNF-a and IL-6 are directly correlated with adiposity and insulin resistance.

With the purpose to investigate apelin expression in patients with essential hypertension and obesity in Ukraine patients. The increased level of peptide apelin in hypertensive patients with visceral type of obesity was detected.

Results: the average means of WC, BMI and apelin level in total group were significantly higher in comparing with control group. Patients were categorized into 4 cluster groups based on k-means according apelin and BMI data. The 1st and 2nd clusters had opposite meanings of BMI. Patients of the 1st cluster had the lowest BMI and also the shortest duration of the disease. No significance were in BMI data between patients of 3rd and 4th clusters, but the opposite apelin activity was detected. In cluster 4, adipokine’s activity was the lowest one from total amount of patients and in cluster 3 – the highest one. Patients of 4th cluster had pronounced carbohydrate disorders and dyslipiemia. Significant correlations of apelin with parameters of carbohydrate pool were found. Analysis of apelin’s interrelations in total group showed significant correlations with parameters of carbohydrate pool. Numerous positive correlations of apelin were found: with fasting insulin (R=0,29, p<0,05), -post OGTT glucose and insulin levels (R=0,39 and R=0,41 respectively, p<0,05), -HOMA index (R=0,24, p<0,05) and HbA1c (R=0,24, p<0,05). In patients of cluster 1 the significant correlation of apelin and HbA1c was estimated (R=0,53, p<0,05). In patients of 2nd and 4th clusters significant negative correlations of apelin with BMI were detected (R=-0,72 and R=-0,41 respectively, p<0,05).

Summary: Correlations of apelin with components of carbohydrate pool proves an influence of apelin on glucometabolic disorders. Different results that concern influence of apelin in insulin resistance, prediabetes, type 2 diabetes, arterial hypertension need further investigations. Strong association of apelin with type 2 diabetes development was found, prediction of cardiovascular complications in patients with metabolic syndrome was analyzed.

Significant dyslipiemia with high atherogene index, dysglicemia, hyperinsulinemia, and pronounced expression of pro-inflammatory cytokine were accompanied with decreasing of apelin level and negative correlation of BMI with peptide. Increasing of plasma apelin level in obesity may play protective role by delaying the development of type 2 diabetes mellitus. Over expression of apelin in hypertensive patients with moderate abnormalities in lipid and carbohydrate metabolism was considered as compensatory reaction. Further investigations of apelin activity will lead to clarifying the potential links of metabolic parameters with peptide expression.