**Metformin and left ventricular hypertrophy in patients with comorbidity**

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Essential hypertension (EH) stays the important public challenge, because of leading positions in morbidity and mortality in not only Ukraine, but also worldwide. Recent studies have suggested that metformin could inhibit cardiomyocyte apoptosis and improve cardiac function. Pathological left ventricular hypertrophy is a crucial pathological condition that triggers several serious cardiac events, including arrhythmias, heart failure, and sudden death. Recent studies have suggested that metformin could inhibit cardiomyocyte apoptosis and improve cardiac function. However, whether metformin has an inhibitory effect on cardiac hypertrophy hasn’t been clarified.

**Aim of the study** was to investigate metformin’s influence on left ventricular structure and function in patients with essential hypertension with concomitant type 2 diabetes (T2D).

**Materials and methods:** 120 patients with essential hypertension (EH) were recruited in the study and were divided into three groups according to comorbid state: 60 - EH and T2D; 30 – EH with prediabetes; 30 – EH without dysglicemia. Carbohydrate parameters, left ventricle structure and function were analyzed before and after 12 weeks of metformin treatment. Metformin was prescribed after titration period in average dosage of 1000 – 2000 mg. Taking in account, that metformin is not allowed for prescription to the patients with prediabetes, such patients received life style modification recommendations. The results were analyzed before and in 12 weeks treatment period. Antihypertensive treatment aim was arterial pressure level ≤ 140/85. Antiglicemic treatment considered as successful in case of HbA1c level ≤ 7 %.

**Results.** Scientific data present that cardioprotective effects of metformin are connected with lipid metabolism enhancing, endothelial function and vessel reactivity improving, hemostasis disorders diminishing. Our previous data showed, metformin treatment results in fasting glycaemia and insulin resistance diminishing on 21,79 % and 26,84 %, lowering abdominal fat deposition for 5,54 %. Improving of endothelial function in metformin treatment is connected with endothelial nitric oxide synthase raising on 8,43%, inducible nitric oxide synthase diminishing on 20,62 %, with nitric oxide bioavalibility enhancing on 36,6 % by decreasing of S-nitrozothiol level. 12 weeks metformin treatment showed positive tendency not only in carbohydrate and lipid parameters with insulin sensitivity and endothelial function improving, but also it has lead to apelin level 33,3 % increasing, vascular endothelial growth factor attenuating on 22,0 %, oncostatin M and interleukin-6 levels diminishing on 18 % and 15 % correspondently.

Echocardiography in 12 weeks metformin treatment showed significant decreasing of left ventricle myocardium mass for 6,1 %, left ventricle posterior wall thickness - 2,3 %. More pronounced changes in patients with EH and T2D are connected with glucotoxicity, lipotoxicity, insulin resistance diminishing and also with pleiotropic metformin’s effects.

**Conclusion.** Metformin has positive influence to the structure and function of left ventricle with increasing of EDV and LV hypertrophy regress. These findings may provide a potential effectiveness for patients with T2D at risk of developing pathological cardiac hypertrophy.

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