Formation of cardio-renal syndrome in patients with chronic heart failure and type 2 diabetes, depending on the choice of glucose-lowering therapy

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Objective: To evaluate the contribution of various glucose-lowering drugs and their combinations to the formation of cardio-renal syndrome in patients with chronic heart failure (CHF) and chronic kidney disease (CKD).

Materials and methods: 223 patients with CHF were examined, among whom 98 patients had a history of type 2 diabetes mellitus. In 67 subjects, CKD was identified. The diagnosis of CHF was confirmed by the presence of myocardial dysfunction according to echocardiography and an increase in the concentration of N-terminal fragment of the brain natriuretic peptide (NT-proBNP) in the blood of more than 125 pg / ml by ELISA. The diagnosis of CKD was confirmed by lowering the estimated glomerular filtration rate using the formula CKD-EPI (eGFR) <60 ml / min / 1.73 m² for at least 3 months.

The filtration function of the kidneys was also determined by the level of cystatin C in the blood and Cystatin-based eGFR (CKD-EPI). Depending on the received sugar-lowering therapy, patients with CHF and CKD were divided into four groups: the first group included 21 patients (31.3%) taking metformin, the second group included 12 (17.9%) patients receiving insulin therapy, the third - 15 (22.4%) surveyed taking sulfonylurea drugs, in the fourth group - 29 (43.3%) people who used a combination of metformin and sulfonylurea drugs.

Results: the groups did not differ by gender, age, comorbidity or concomitant therapy, as well as in type of CHF depending on LV EF (for rLVEF pmsg=0,124; for mrlVEF pmsg =0,459; and for pLVEF pmsg =0,691). The average LV EF was, respectively, 52,3±7,8%, 56,0±9,2%, 51,8±6,0%, 58,5±9,1% (pmsg =0,731).

NT-proBNP was significantly different between the groups: the maximum in the second group was 687.5±129.4 pg/ml, the minimum in the first group was 209.4±96.5 pg/ml, the intermediate value in the third and fourth group, respectively: 348.6±87.7 pg / ml and 367.9±99.7 pg / ml (pmsg =0,002).

Parameters of the LV diastolic function were significantly different between groups: septal e’, lateral e’, E/e’ in the second group were higher than in the first, third and fourth group (pmsg <0,001 for all parameters). Groups did not statistically significantly differ in Creatinine-based eGFR (pmsg = 0.216). Cystatin C and Cystatin-based eGFR did not depend on the choice of glucose-lowering therapy but correlated with NT-proBNP (r = 0.685, p <0.05 and r = -0.804, p <0.05, respectively). Conclusion: in patients with type 2 diabetes and CKD, the type of CHF does not depend on the choice of glucose-lowering therapy. In patients who take insulin therapy, more severe CHF is formed, assessed by NT-proBNP, and while taking metformin is less pronounced. The use of Cystatin-based eGFR (but not Creatinine-based eGFR) to assess the dynamics of CKD showed that the decrease in filtration function of the kidneys is not associated with the choice of glucose-lowering drugs, but depends on the CHF severity.