PREVENTION OF THE PROGRESSION OF CHRONIC KIDNEY DISEASE BY DECOMPENSATION OF CHRONIC HEART FAILURE

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Annotation.

The progression of chronic kidney disease in patients with acute decompensation of chronic heart failure is associated with a complex of causes. In acute decompensation of chronic heart failure with a decrease in the left ventricular ejection fraction, there is a decrease in renal perfusion, which leads to a subsequent decrease in the glomerular filtration rate (GFR) [1]. Another possible cause may be adverse reactions of the body to aggressive therapy with loop diuretics, which is part of the complex therapy for acute decompensation of chronic heart failure [2]. Such adverse reactions include: activation of neurohormonal systems, leading to increased resistance of the renal vessels, as well as hypovolemia and electrolyte disturbances, leading to ischemia and hyperosmolar damage to the renal tubules [3, 4]. The progression of chronic kidney disease is an independent risk factor for the development of cardiovascular complications and the cause of death in patients is 10-20 times more common than in the general population [5]. Prevention of the progression of chronic kidney disease in patients with acute decompensation of chronic heart failure can be effective in improving kidney perfusion by reducing the resistance of the organ's vessels and reducing the adverse reactions of loop diuretic therapy. Improvement of renal perfusion is possible with the addition of standard therapy for acute decompensation of chronic heart failure with dihydropyridine-type calcium antagonists. These drugs can achieve a pronounced renoprotective effect in patients with diseases of the cardiovascular system and diabetic nephropathy by restoring the vasomotor tone of the afferent arterioles of the renal glomeruli [6, 7]. Reducing the adverse reactions of loop diuretic therapy is possible when changing the drug administration regimen from single bolus injections to extended intravenous infusion [8, 9].

Keywords:

Glomerular filtration rate, hypertension disease, the functional state of kidneys, renal hemodynamics

Materials and methods

We examined 125 patients (men — 56, women — 69) admitted to the therapeutic department with acute decompensation of chronic heart failure. The median age of the patients was 76 (74; 79) years. The diagnosis of acute decompensation of chronic heart failure was established on the basis of at least one symptom (suffocation, orthopnea, edema) and one clinical sign of chronic heart failure (wheezing in the lungs, peripheral edema, enlarged liver, ascites, congestion in the lungs on the X-ray,

swelling of the cervical veins). An additional inclusion criterion was a history of chronic heart failure. Not included patients with kidney disease, accompanied by severe structural adjustment (chronic pyelo and glomerulonephritis, polycystic disease and congenital anomalies of the kidneys, hydronephrosis), the 5th stage chronic kidney disease (end-stage renal failure), acute infectious or inflammatory diseases, systolic blood pressure below 100 mm. Hg.St. and the need for intravenous inotropic use means, except digoxin. Also, the study did not include patients with acute coronary syndrome or stroke suffered in the last 6 months. The following causes of acute decompensation of chronic heart failure were established: non-compliance with the water-salt regime in 37 (29.6%), non-compliance with the drug regimen (Angiotensin converting enzyme inhibitors, beta-blockers) in 54 (43.2%), a combination of these causes in 34 (27.2%). The cause of chronic heart failure in all was coronary heart disease. The median duration of chronic heart failure was 7 (6; 8) years. Concomitant pathology was distributed as follows: arterial hypertension in 76.8%, a history of myocardial infarction in 61.6%, chronic obstructive pulmonary disease in 34.4%, atrial fibrillation in 31.2%, diabetes mellitus in 24.8%, obesity (body mass index greater than 35 kg/m^2) in 32.8% of patients. All patients underwent a standard clinical examination. The state of renal function was monitored by two methods: by the level of serum creatinine (sCr) in mmol / l and by the level of cystatin C in mg/l. The study of sCr and cystatin C, as well as the assessment of $GFR_{creatinine}$ and $GFR_{cystatin\ C}$, was carried out at the following stages: on admission (stage 1), on day 10. hospitalization (stage 2). All patients were diagnosed with chronic kidney disease of different stages based on the baseline sCr and GFRcr levels. The criteria for chronic kidney disease were: an increase in sCr relative to "basal" values, a decrease in GFRcr, and a change in urinary sediment. 39 patients (31.2%) had stage II CKD, 32 — stage III, 33 — stage III, and 21 — stage IV. For the progression of chronic kidney disease, a decrease in GFR was taken when calculated for any of the indicators (or for two indicators at the same time), which transferred the patient to a more severe stage. The exception was patients with stage IV CKD who did not have cases of transition to a more severe stage, but an increase in creatinine levels by 50% or more corresponded to the risk of developing acute renal damage.

The patients were divided into 2 groups. Group 1 (n — 60) included patients who were treated with conventional therapy for CHD: Angiotensin-converting enzyme inhibitors, beta-blockers, loop diuretics, and mineralocorticoid receptor antagonists, if necessary, cardiac glycosides and potassium preparations. In group 2 (n-65), therapy was supplemented with a program to prevent the progression of chronic kidney disease. The program included an extended intravenous infusion of furosemide using an intravenous infusion pump. The daily dose was divided into two injections, the rate of one administration was 20 mg / hour. The daily dose varied depending on the balance of the secreted fluid over the injected one and the rate of diuresis. The total dose of furosemide administered intravenously during the course of therapy was 3.53 (2.06; 4.00) mg/kg. The groups did not differ in age, gender, the number of patients with different stages of CKD, the severity of the clinical condition of acute decompensation of chronic heart failure according to the clinical condition assessment scale, left ventricular ejection fraction (LV EF), and GFR. The duration of parenteral therapy with diuretics and their total dose in the groups did not significantly differ.

Results

At the 1st stage of the study, 14 patients of both groups showed progression of chronic kidney disease, which was manifested by a significant decrease in GFR by two indicators. The sCr level increased by 32.1% (p=0.002), the SCFcr decreased by 37.5% (p=0.009). The median cystatin C index was 2.45 mg/L, and the median GFR was 29 ml/min/1.73m^2. Relative to the initial GFR indicator, determined by the sCr level, the GFR_{cist} decreased by 39.5% (p=0.011). There were no significant differences between GFRcr and GFRcist at the 1st stage of the study. At the 2nd stage, a total of 24 patients were identified with signs of progression of chronic kidney disease in two parameters. There were no significant differences between the level of the studied indicators at the 1st and 2nd stages of the study. GFRkr and GFRcist were the same at the 2nd stage. An isolated decrease in GFRcist at the 1st stage of the study was found in 25 patients. Median cystatin C score was 2.34 mg/l, and the median GFR was 31 ml/min/1.73 m2. Relative to the baseline GFRcr, GFRcist was significantly lower

by 36.7%. At the same stage of the study, GFRcist was 39.2% lower than GFRcr (p<0.001). At the 2nd stage, an isolated decrease in GFRcist was found in 13 patients. The median cystatin C index was 2.38mg/L, and the median GFR was 29ml/min/1.73 m2. Thus, the GFRcist decreased by 40.8% relative to the initial GFR index (p=0.011), and was significantly lower than the GFRcr of the 2nd stage of the study by 46.3%. In group 1 (n — 60), upon admission to the hospital, we identified 20 cases of progression of chronic kidney disease (33.3%). In 6 patients (10.0%), this was manifested by a change in GFR by two indicators, in 14 (23.3%), in isolation by serum cystatin C. Among the patients of the first group with the Progression of chronic kidney disease at the 2nd stage of the study, 1 (1.7%) case of recovery of two previously reduced GFRcr and GFR cist, and 1 (1.7%) case of recovery of isolated reduced GFRcist to the initial level without worsening of GFRcr was established. No positive dynamics was observed in 5 patients with two low indicators. In the remaining 13 patients (22.8%), who were admitted with an isolated decrease in GFRcist at the 2nd stage of progression of chronic kidney disease, a significant change in GFRcr was confirmed. The total number of episodes of negative dynamics of GFR for two indicators in group 1 was 18 people. There were no significant differences between the indicators of GFRcr and GFRcist in this category of patients at the 2nd stage of the study. At the same time, there were 7 new cases of progression of chronic kidney disease (12.3%), manifested by isolated changes in GFRcist, and 2 new cases of isolated decrease in GFRcr (3.5%). Thus, the total number of progression of chronic kidney disease among patients of the 1st group at the 2nd stage of the study was 27 (47.4%). Including two indicators of 18 (31.6%), isolated by GFRcist - 7 (12.3%) according to GFRcr — 2 (3.5%). In 13 cases, the progression of chronic kidney disease was diagnosed at an early stage by a change in the level of cystatin C and later confirmed by a change in sCr. In group 2 (n — 65), upon admission to the hospital, we identified 19 cases of progression of chronic kidney disease (29.3%). In 8 patients (12.3%), there was a decrease in GFR by two indicators, in 11 (17.9%) by serum cystatin C. At the 2nd stage of the study, in 4 patients (6.2%) with an isolated decrease in GFRcist, the indicator was restored to the initial level without worsening of GFRcr. In two cases (3.1%), both previously changed indicators were restored to the initial level. In 6 similar cases, there were no significant changes from the previous stage. In 7 patients with an isolated decrease in GFRcist at admission, at the 2nd stage of progression of chronic kidney disease, a change in GFRcr was confirmed. At the same time 2 new cases of chronic kidney disease progression were identified: 1 (1.6%) for the GFRcis and 1 (1.6%) for the GFRcr. The total number of cases of progression of chronic kidney disease in group 2 at the 2nd stage of the study was 15 (23.4%). Including two indicators 6 (9.4%) and GFRcist — 9 (14.1%). In 7 cases (10.8%), the progression of chronic kidney disease was diagnosed at an early stage by a change in the level of cystatin C and later confirmed by a change in sCr.

Discussion

At admission to the hospital, there were no significant differences between the groups. In group 1, renal dysfunction was observed in 33.3% of patients, in group 2 in 29.3%. The causes of these disorders were acute decompensation of chronic heart failure. On day 10, in group 1, the number of progressions of chronic kidney disease increased to 47.4% due to the appearance of new cases that were detected by monitoring GFR for serum cystatin C levels. New cases of deterioration of renal function were observed in 15.0% of patients of group 1. In group 2, at this stage, the progression of chronic kidney disease did not significantly change compared to the previous stage, and was observed in 23.4% of patients. New cases of impaired renal function at this stage were found in 3.1% of patients. Comparison of the number of new cases of chronic kidney disease progression in groups on day 10. it was shown that in the 2nd group there were 4.8 times less of them (p<0.05). Restoration of previously reduced renal function in group 1 on day 10. it occurred in 3.3% of patients, in group 2 in 9.2%. Comparative analysis showed that the differences in the number of recoveries in the groups were unreliable. Thus, the prevention program has reduced the number of new cases of kidney function disorders during the treatment of acute decompensation of chronic heart failure and thereby reduce the total number of episodes of progression of chronic kidney disease on day 10. The program had no effect on the number of kidney function recoveries.

Conclusion

The prevention program, including the use of the calcium channel antagonist nitrendipine and the replacement of single bolus injections of furosemide with extended intravenous infusion in the complex of therapy for acute decompensation of chronic heart failure, significantly reduced the number of cases of progression of chronic kidney disease by 2.0 times. This is due to a 4.8-fold decrease in the number of new episodes of renal dysfunction that occur during the treatment of acute decompensation of chronic heart failure.

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