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RISK FACTORS FOR THE DEVELOPMENT OF CKD IN PATIENTS WITH CORONARY ARTERY DISEASE

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RESUME

Purpose of the study: to determine the risk factors for the development of CKD in patients with coronary artery disease (CAD) after myocardial revaslarization. **Material and methods.** The study included 160 patients with coronary artery disease who underwent coronary angiography. The study included patients with eGFR over 60 ml / min. On the second day after the endovascular procedure, the patients underwent determination of the blood creatinine concentration to identify patients who developed contrast-induced nephropathy (CIN). In dynamics, at the end of the second year of follow-up all patients underwent repeated examination. **Research results**. CIN in the early period after endovascular intervention was observed in 37 patients. All patients with CAD included in the study were distributed depending on the eGFR achieved by the end of the 2nd year after revascularization. In 20% (32 out of 160) patients, an eGFR of less than 30 ml / min was achieved. Comparative analysis of the parameters revealed that patients with the worst prognosis in terms of the development of CKD were significantly older ($p < 0.01$), they initially had more impaired renal filtration function ($p < 0.001$). **Conclusion.** The most significant prognostic marker for the development of CKD IV-V in patients with coronary artery disease within 2 years after revascularization is the development of contrast-induced nephropathy in the early period after endovascular intervention (increases the risk of CKD IV-V by 7.31), the concentration of BNP in the peripheral blood serum is more than 587 pg / ml (increases the risk of CKD IV -V 10.16 times), LVEF less than 52% (increases the risk 9.72 times) and the sphericity index more than 0.62 units (increases the risk 8.77 times).

KEYWORDS: chronic kidney disease, ischemic heart disease, contrast-induced nephropathy, echocardiography.

There are common factors in the pathogenesis of the development of cardiovascular and renal diseases, such as neurohumoral activation and atherosclerotic vascular lesions.[1] The combination of CKD and coronary artery disease and chronic heart failure (CHF) significantly worsens the prognosis and quality of life of patients. CKD is the most important risk factor for cardiovascular events, and with an increase in the stage of CKD, the cardiovascular risk increases.[2,3] CHF itself is a syndrome associated with a high risk of hospitalization and mortality. The combination of these pathologies in various variants is defined as cardiorenal syndrome 4,[5] According to the data presented, a diagnostic panel, including various biomarkers of both cardiac and renal damage, can be used to diagnose type II cardiorenal syndrome.[6,7]

Purpose of the study: to determine the risk factors for the development of CKD in patients with coronary artery disease after myocardial revaslarization.

MATERIAL AND METHODS

The study included 160 patients with coronary artery disease who underwent coronary angiography. The average age of the patients was 56.6 ± 1.27 years. The study included patients with eGFR over 60 ml / min. All patients were prescribed standard CHD therapy (antiplatelet agent - aspirin or clopidogrel, and in the case of endovascular revascularization - dual antiplatelet therapy; beta-blocker, acetyl-CoA reductase inhibitor - atorvastatin, RAAS blocker - valsartan). On the second day after the endovascular procedure all patients underwent repeated determination of the blood creatinine concentration to identify patients who developed contrast-induced nephropathy (CIN). CIN was determined in the case of an increase in creatinine by 25% or more from the initial level.

In dynamics, at the end of the second year of follow-up after revascularization, all patients underwent a second examination to determine the prognostic significance of the studied parameters in terms of the development of CKD.

Echocardiographic examination (EchoCG) using an ultrasound probe with a frequency of 2.5-5 MHz and using standard approaches and positions. The following parameters were recorded: LV end-diastolic volume (LV EDV) and LV end-systolic volume (LV ESV) calculated

by the modified Simpson's method; the volume of the left atrium (LA); end systolic and diastolic dimensions of the LV; the thickness of the interventricular septum and the posterior wall of the left ventricle in diastole and systole; the diameter of the basal part of the LV cavity and the length of the LV.

After registration of these indicators, the following indicators were calculated: LV myocardial mass, indexed to body surface area (LVMMI); LV ejection fraction. The regional contractility of the LV and RV was assessed visually using a 17-segment model of the LV (each segment according to a 4-point system: 1 - normokinesis, 2 - hypokinesis, 3 - akinesis, 4 - dyskinesis) and the regional contractility disorder index was calculated: INRS = ∑ points by segment / number of segments assessed. Spectral tissue Doppler sonography of the lateral edge of the mitral annulus fibrosus was performed, at which the maximum rates of diastolic displacement of the mitral annulus fibrosus in the phase of early and atrial LV filling (E and A) and their ratio were recorded. EDV of the right ventricle (RV), EDV of the RV: planimetric method, apical position, 4-chamber projection. Subsequently, the indicator was indexed to the area of the body surface (iPS). The fraction of RV area reduction (RV FUP) was calculated using the formula: RV FUP = (RV end diastolic area - RV end systolic area) / RV end diastolic area * 100%.

The concentration of brain natriuretic peptide (BNP) was estimated by the concentration of the N-terminal fragment of the precursor peptide BNP by a quantitative immunological method in heparinized venous blood. (Cobas h 232 apparatus with appropriate test strips, Roche Diagnostic, measuring range 60-3000 pg / ml). To assess the functional state of the kidneys, the blood creatinine concentration was determined with the calculation of eGFR (Hojs R et al. Clin Nephrol. 2008).

All indicators obtained in the study were entered into the pivot tables of the Excel editor for Windows 2007, grouped according to the studied characteristics and, after checking the normal distribution, were generalized using the arithmetic mean values and the standard deviation of the arithmetic means. The reliability of intergroup comparisons was carried out using the Student's test for paired and unpaired differences.

RESULT AND DISCUSSION

On the second day after the endovascular procedure (CAG / PCI), all patients underwent repeated determination of the blood creatinine concentration to identify patients who developed contrast-induced nephropathy (CIN). CIN in the early period after endovascular intervention was observed in 37 patients. CIN treatment was carried out by the method of hydration, according to the ERBP recommendations [8]. All patients with CAD included in the study were distributed depending on the eGFR achieved by the end of the 2nd year after revascularization. So in 20% (32 patients out of 160) patients achieved eGFR less than 30 ml / min. A comparative analysis was carried out to identify characteristics that differ in patients with eGFR by the end of the second year of observation above and below 30 ml / min. It was revealed that patients with a worse prognosis in terms of CKD development were significantly older ($p < 0.01$), they had a more impaired renal filtration function at baseline ($p < 0.001$). Also, these patients were distinguished by more pronounced structural and functional remodeling of the heart, which was manifested by dilatation of all heart chambers ($p < 0.01$), LV hypertrophy ($p < 0.001$), decreased systolic function ($p < 0.001$) and a tendency to restrictive LV diastolic dysfunction ($p < 0.001$) and an increase in Tei ($p < 0.001$), an increase in BNP concentration ($p < 0.001$) and pulmonary hypertension ($p < 0.001$).

At the heart of the violation of glomerular hemodynamics is an increase in venous pressure. A reflection of this process is myocardial hyperextension, in response to which the ventricular cardiomyocytes express sodium uretic peptide (NPP). An increased concentration of NP in the peripheral blood may be a predictor of the progression of CKD in patients with CHF. Trail studies conducted in different years (Kim HN, Januzzi JL, 2011, Balion C., Don-Wauchope A., 2013, Volpe M.2014) have shown that the NPL level independently predicts the risk of hospitalization, death from all causes in patients with acute decompensation of CHF. Based on the differences found, we analyzed the predictive significance of prognostic markers for the development of CKD IV-V in patients with CAD within 2 years after revascularization (Table 2).

Table 1. Comparative characteristics of patients with CAD patients, depending on the eGFR achieved by the end of the observation period.

	eGFR by the end of 2 years less than 30 ml / min	eGFR by the end of 2 years 30 ml / min or more
Age, years	59,16±1,60	54,29±0,73**
eGFR, ml / min	92,12±2,65	105,84±1,62***
Creatinine, μmol / L	100,97±2,87	87,45±1,81***
MNUP, pg / ml	1166,81±123,90	591,80±69,48***
uric acid, mmol / l	4,89±0,17	4,96±0,09
GARDEN, mm Hg	120,00±6,37	124,38±2,04
DBP, mm Hg	78,44±4,56	80,23±1,98
Heart rate, beats per	103,84±4,93	94,84±2,27

minute		
ICDO, ml / m2	93,09±3,56	81,37±1,35**
ILP, ml / m2	60,31±2,75	51,78±1,55**
IPP, cm2 / m2	12,97±0,55	11,21±0,24**
iPZh, cm2 / m2	20,31±0,67	18,61±0,34*
MVP, mm	10,31±0,29	10,65±0,12
ZSLZh, mm	9,94±0,25	10,33±0,14
LVMMI, g / m2	166,63±5,68	136,47±3,32***
ind of spheres, rel.	0,77±0,02	0,65±0,01***
LVEF,%	37,56±2,30	51,63±0,49***
INRS, score	1,98±0,09	1,44±0,03***
FUP PZh,%	34,41±1,06	35,43±0,74
UI, mo / m2	32,90±1,61	41,88±0,77***
MI, ml / m2	3424,22±254,59	4003,36±134,25*
Tei LV, rel.	0,63±0,02	0,49±0,01***
Tei PZh, rel.	0,53±0,02	0,51±0,01
avg R LA, mm Hg	27,91±0,95	23,02±0,39***

*Note: * - reliability of differences between groups. One sign - p <0.05, two signs - p <0.01, three signs - p <0.001.*

Table 2.Prognostic markers of the development of CKD IV-V in patients with coronary artery disease within 2 years after revascularization and their predictor significance.

Predictive marker (median used)	Patients at risk of developing CKD IV-V in the presence of a marker (%)	Patients at risk of developing CKD IV-V in the absence of a marker (%)	Chi square (reliability of the criterion)	The relative risk of developing CKD IV-V in the presence of a marker
Over 56 years old	18/76 (23,68%)	14/84 (16,67%)	1,25 (нд)	
KIN +	22/37 (59,46%)	10/123 (8,13%)	45,86 (p<0,001)	7,31
eGFR initially less than 102.8 ml / min	26/80 (32,50%)	6/80 (7,50%)	15,66 (p<0,001)	4,33
MNUP more than 587 pg / ml	29/78 (37,18%)	3/82 (3,66%)	28,08 (p<0,001)	10,16
ICDO LV more than 83ml / m2	21/80 (26,25%)	11/80 (13,75%)	3,95 (p<0,05)	1,91
ILP more than 49ml / m2	21/78 (26,92%)	11/82 (13,41%)	4,59 (p<0,05)	2,01
IPP more than 12cm2 / m2	18/56 (32,14%)	14/104 (13,46%)	7,77 (p<0,01)	2,39
iPZh more than 19 cm2 / m2	17/46 (36,96%)	15/114 (13,16%)	11,27 (p<0,001)	2,81
LVMI more than 150.5 g / m2	26/80 (32,5%)	6/80 (7,5%)	15,66 (p<0,001)	4,33
Sphericity index more than 0.62 units	28/71 (39,44%)	4/89 (4,49%)	30,03 (p<0,001)	8,77
LVEF less than 52%	28/67 (41,79%)	4/93 (4,30%)	34,02 (p<0,001)	9,72
INRS more than 1.28 points	26/76 (34,21%)	6/84 (7,14%)	18,26 (p<0,001)	4,79
DDLZH type 3	18/37 (48,65%)	10/92 (10,87%)	21,68 (p<0,001)	4,48
Tei LV more than 0.51 rel units	24/64 (37,50%)	4/65 (6,15%)	18,68 (p<0,001)	6,09
Aircraft Rav more than 24mm Hg	23/75 (30,67%)	9/85 (10,59%)	10,03 (p<0,01)	2,90

CONCLUSION

The most significant prognostic marker for the development of CKD IV-V in patients with coronary artery disease within 2 years after revascularization is the development of contrast-induced nephropathy in the early period after endovascular intervention (increases the risk of CKD IV-V by 7.31), the concentration of BNP in the peripheral blood serum is more than 587 pg / ml (increases the risk of CKD IV -V 10.16 times), LVEF less than 52% (increases the risk 9.72 times) and the sphericity index more than 0.62 units (increases the risk 8.77 times).

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