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Research Article

Approaches to therapy Amlodipine/Indapamide/ Perindopril therapy of high arterial hypertension in ischemic heart disease patients with chronic kidney disease stage 1-3 after coronary stenting

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Abstract

Introduction: Acute coronary syndrome, chronic forms of ischemic heart disease (IHD) and postinfarction cardiosclerosis are the main causes of morbidity and mortality in the world, including Russia. In the XXI century, there is an increase in comorbid pathology, especially in the combination of IHD with arterial hypertension, diabetes, chronic obstructive pulmonary disease and chronic kidney disease. Patients with IHD and chronic kidney disease have a higher incidence of coronary events and complications. The frequency of coronary events and complications indicates the need to improve the diagnosis and treatment of this group of patients. diagnosis and treatment of this group of patients. **The aim of the study was** to analyze the dynamics of vascular stiffness, pulmonary hypertension (PH), diastolic heart dysfunction and endothelial dysfunction indices in patients with different variants of ischemic heart disease combined with chronic kidney disease (CKD) stage 1-3 using complex therapy of combined hypotensive drug Amlodipine/ Indapamide/Perindopril three months after coronary stenting and to compare them with the group of patients on conservative therapy only.

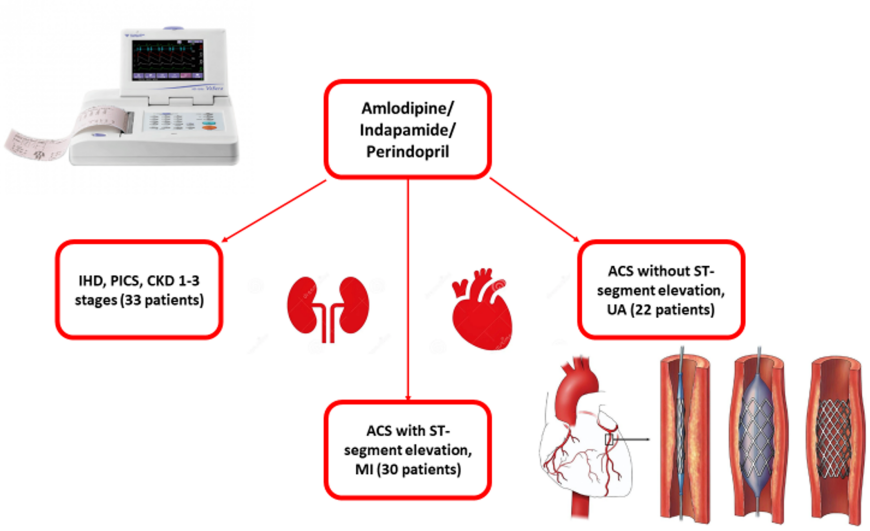
Material and Methods: 85 patients with different forms of IHD, arterial hypertension (AH) on the background of CKD 1-3 stages, as well as data of 42 patients with IHD, AH without renal pathology were analyzed. The first group – IHD, postinfarction cardiosclerosis, CKD stage 1-3 (33 patients); the second group – acute coronary syndrome with ST-segment elevation, Myocardial infarction (MI) (30 patients); the third group – ACS without ST-segment elevation, Unstable angina (UA) (22 patients).

Results: The highest indices of vascular stiffness (Pulse wave velocity (PWV), Augmentation index (AI), CAVI, Central Systolic Blood Pressure (SBPao), central arterial pulse pressure (PP)) were registered in combination of ACS with ST-segment elevation and CKD 1-3 stages. These indices are markers of IHD progression in these patients; they also have increased pulmonary hypertension and diastolic dysfunction of the heart, endothelial dysfunction with vasodilation insufficiency in 88% of cases, which even without hemodynamically significant coronary artery stenoses according to coronary CT angiography data leads to the development of ACS with ST-segment elevation and ACS without ST-segment elevation with MI.

Amlodipine/Indapamide/Perindopril was prescribed to all patients due to high arterial hypertension on admission against the background of basic therapy of IHD and coronary stenting. Coronary CT angiography in patients with comorbid renal pathology does not lead to aggravation of chronic kidney disease after 3 months, on the contrary; in this group of patients the most pronounced decrease of arterial stiffness (AS), AI, SBPao, PP with elevation of glomerular filtration rate (GFR) and decrease of creatinine in blood occurs in comparison with the group of patients who did not undergo coronary stenting, they were only on conservative therapy.

Conclusion: Prescription of three component drug Amlodipine/Indapamide/Perindopril on the background of baseline therapy especially in combination with surgical vascularization of the heart is justified.

Graphical abstract



Keywords

coronary heart disease, chronic kidney disease, postinfarction cardiosclerosis, unstable angina pectoris, acute coronary syndrome, coronary stenting, amlodipine, indapamide, perindopril

Introduction

Acute coronary syndrome, chronic forms of IHD, postinfarction cardiosclerosis are ranged first among the causes of morbidity and mortality in many countries of the world, including Russia. The 21st century is characterised by a rapid growth of comorbid pathology, especially when combining various variants of IHD with arterial hypertension (AH), diabetes, chronic obstructive pulmonary disease (COPD), chronic kidney disease (CKD).

Thus, according to the literature, the incidence of IHD in patients with CKD is higher by 22-30%, and new coronary events and complications are registered 3.4 times higher compared to IHD patients with preserved renal function (Agarwal et al. 2021; Bergmark et al. 2022). If the therapy of IHD is developed in detail, especially taking into consideration modern cardiac surgical methods of treatment, the treatment of IHD patients with comorbid renal pathology is not sufficiently covered in the literature, which leads to difficulties in diagnosis and treatment of this cohort of patients in the clinical practice (Zhuravleva et al. 2020; Karpov et al. 2020; Boncevich et al. 2021, 2022; Nadirova et al. 2023).

The search for new modern methods of examination and the effect of combined hypotensive drugs on the main links in the pathogenesis of IHD and CKD remains relevant, which allows reducing the number of complications and mortality from this frequent comorbid pathology, especially in emergency cardiology (Tsygankova et al. 2020; Chaulin et al. 2020; Safronenko et al. 2021; Pribylov et al. 2022; Ebzeyeva et al. 2023). According to the latest Russian clinical recommendations (Kobalava et al. 2020) for the treatment of arterial hypertension, it is necessary to prescribe combined hypotensive drugs with fixed dosages from the first stage.

Our earlier studies proved the positive effect of individual drugs: Angiotensin-converting enzyme inhibitors (iACE), Angiotensin-II receptor blockers (ARBs/sartans), Calcium channel blockers, Thiazide and thiazide-like diuretics, especially when using coronary

angioplasty on the main pathogenetic mechanisms of IHD and CKD with improvement of endothelial, diastolic dysfunction, reduction of vascular wall stiffness (Brouwers et al. 2021; Bhatt et al. 2022; Pribylov et al. 2022).

In the light of recent guidelines on the use of combined hypotensive drugs for the treatment of comorbid pathology of IHD with AH and CKD, it seems relevant to estimate the effects of Amlodipine/Indapamide/Perindopril on the background of complex etiopathogenetic therapy, as well as to analyse the efficacy of coronary stenting in patients with various forms of IHD on the background of CKD stage 1-3 during hospital rehabilitation, then after 12 weeks of outpatient treatment (Hisatome et al. 2021; Hoeper et al. 2022; Byrne et al. 2023).

The aim of the present study: to analyse the dynamics of vascular wall stiffness, pulmonary hypertension, diastolic dysfunction of the heart and endothelium in patients with chronic and acute variants of IHD on the background of CKD stage 1-3 using the combined hypotensive drug Amlodipine/Indapamide/Perindopril in complex therapy 3 months after coronary stenting.

Materials and Methods

Objects under study

The study was conducted at the Department of Internal Medicine of Kursk State Medical University (KSMU) on the basis of Kursk Regional Multidisciplinary Clinical Hospital, agreed with the regional ethical committee, minutes №8 of 18.10.2022. Eighty-five patients with various forms of IHD (ACS with ST-segment elevation, ACS without ST-segment elevation, MI, PICS) in combination with AH and CKD stage 1-3, as well as 42 patients with IHD, AH with preserved renal function (control group) were studied. The duration of outpatient follow-up after hospital discharge was 12 weeks. The study was approved by the local ethical committee. The criteria for verification of IHD were: a history of MI, a positive result of non-invasive stress test for detection of coronary insufficiency, coronary angiography at admission of patients to the regional vascular center. The diagnosis of CKD was established in these patients on an outpatient basis 3-8 years before hospitalisation, and then verified by calculation of GFR by CKD-EPI and detection of renal damage markers in serum. The genesis of CKD was represented by hypertensive nephropathy. All patients were divided into 3 groups: the first group – IHD, PICS, CKD 1-3 stages (33 patients), the second group – ACS with ST-segment elevation, MI (30 patients), and the third group – ACS without ST-segment elevation, UA (22 patients).

Research methods

Regional arterial stiffness was assessed using volumetric sphygmography on a VS-1500 Fukuda Denshi (Japan) device with determination of Pulse wave velocity (PWV), CAVI, AI, Systolic blood pressure (SBP), Diastolic blood pressure (DBP), central pulse arterial pressure (cPAP); the measurement was performed with simultaneous recording of plethysmography on four limbs, ECG, phonocardiogram, and femoral artery pulse. The studies were performed after CAG in the Regional Vascular Center according to the standards and requirements (Al Ghorani et al. 2024). Assessment of LV diastolic function was performed by two-dimensional echocardiography (Linde et al. 2020).

The degree of endothelial dysfunction was assessed by ultrasound with determination of flow-dependent vasodilation of the brachial artery, in the test with post-occlusive hyperaemia. The endothelin-1 content was determined by immunoenzyme method using Endoteli ” Biomedica” (Austria) reagent kit. Nitric oxide was determined using the Gris reagent (Ott et al. 2022).

Statistical analysis

Statistical processing of the data was performed using Statistica-10 application software package (StatSoft, USA). The value is presented as mean value and standard deviation (M±SD); parametric T-Student criteria were calculated for analysis. The non-parametric Wilcoxon test was used for the related samples and Mann-Whitney test for the unrelated samples. The validity of the data was assessed using the table of critical values; differences were considered statistically significant with $p<0.05$.

Results and Discussion

IHD patients (ACS with ST-segment elevation, ACS without ST-segment elevation, MI) combined with stage 1-3 CKD and a group of IHD patients with preserved renal function were subjected to CAG and investigations of vascular wall stiffness, diastolic dysfunction of the heart, and pulmonary hypertension upon admission to the regional vascular center. Three groups of patients were selected among the examined patients to achieve the aim and objectives of the study: the first group – IHD, PICS, with high AH and CKD 1-3 stages; the second group – ACS with ST-segment elevation, MI, CKD 1-3 stages; the third group – ACS without ST-segment elevation, UA, CKD 1-3 stages, and the comparison group consisted of IHD, PICS patients with AH without renal pathology. Indices of arterial stiffness, diastolic cardiac dysfunction and pulmonary hypertension in IHD patients with high arterial hypertension on the background of chronic kidney disease stage 1-3 in comparison with the group of IHD patients, arterial hypertension with preserved renal function are presented in Table 1.

According to the recent literature, researchers believe that PWV, augmentation index, cPAP may be indicators of cardiovascular complications risk and mortality in patients with IHD (Waterbury et al. 2020). According to our data, PWV, augmentation index were maximally high in patients with ACS with ST-segment elevation and MI, ACS without ST-segment elevation and UA in combination with CKD stage 1-3. PWV and augmentation index, as correctly, are markers of CKD progression (Pribylov et al. 2022).

Our studies also indicate that the main indices of arterial stiffness in IHD patients in combination with high SBP, DBP, SBPao, especially cPAP, are signs of CKD progression and indicators of risk of cardiovascular complications in patients with CKD. Moreover, at the onset of ACS with ST- segment elevation, the maximum systolic pressure in the left atrium increases (up to 40.2±3.3 mmHg) and marked diastolic dysfunction of the heart is detected. E/A ratio decreases to 0.58±0.04 in ACS

Table 1. Indices of arterial stiffness, diastolic cardiac dysfunction and pulmonary hypertension in IHD patients with high arterial hypertension on the background of chronic kidney disease stage 1-3 in comparison with the group of IHD patients, arterial hypertension with preserved renal function

Indices	IHD, AH on the background of CKD stage 1-3			IHD, AH without renal pathology (n=42)
	IHD, PICS(n=33)	ACS with ST-segment elevation, MI (n=30)	ACS without ST-segment elevation, UA (n=22))	
Age, years	58±5.8	56±4.2	50±2.1	59±6.3
BMI, kg/m^2	29.5±4.7*	25.8±3.2*	26.1±4.2*	22.4±2.1
Creatinine, mmol/L	132±12*	128±11*	118±8.4*	88±12
GFR, mmol/min/ 1.73 m^2	50±6.4*	56±3.2*	58±4.2*	98±3.4
Cho, mmol/L	6.9±1.3*	6.4 ± 1.7*	6.7±1.5*	5.6 ±1.2
TG, mmol/L	1.9±0.6	1.8 ±0.8	1.7±0.5	2.1 ±1.1
LDL, mmol/L	4.92±1.2*	4.21±1.5*	3.88±2.2	3.82±1.1
Apo-B lipoproteins, mg/dL	146±8.4*	148±9.1*	134±7.2*	111±9.2
SBP, mmHg	195±28*	188±21*	180±22*	135±10.1
DBP, mmHg	108±11*	100±12*	103±12*	91±8
PWV, m/s	12.13±0.14*	12.08±0.12*	13.1±0.16*	8.97±0.20
AI	1.28±0.11*	1.68±0.13*	1.66±0.11*	1.11±0.12
SBPao, mmHg	172±20.2*	168±14.4*	172±8.6*	128±11.2
cPAP, mmHg	49±10.1*	48±8.0*	51±8.1*	37±6.0
R – CAVI	10.08±0.6*	9.98±0.4*	11.12±0.5*	8.32±0.16
L – CAVI	12.07±0.5*	9.82±0.3*	11.83±0.4*	8.31±0.2
R – ABI	1.16±0.08*	1.18±0.07*	1.18±0.09*	1.15±0.07
L – ABI	1.16± 0.05*	1.15±0.04*	1.19±0.04*	1.10±0.03
E/A	0.68±0.08*	0.58±0.04*	0.68±0.08*	0.72±0.04
PASP, mmHg	38.4±2.2*	40.2±3.3*	39.8±4.2*	29.2±1.8

Note: data are presented as M±SD, * – statistically significant differences between groups ($p<0.001$); ***Abbreviations:*** BMI – body mass index, GFR – glomerular filtration rate, Cho – serum total cholesterol level, TG – triglycerides, LDL – low-density lipoproteins, SBP – systolic blood pressure, DBP – diastolic blood pressure, PWV – pulse wave velocity, AI – augmentation index, SBPao – central systolic blood pressure, cPAP – central pulse arterial pressure, PASP – pulmonary artery systolic pressure.

with ST-segment elevation and MI, in ACS without ST-segment elevation and UA E/A depression up to 0.68±0.08. In patients with acute forms of IHD on the background of CKD stage 1-3, a significant endothelial dysfunction is revealed with vasodilatation insufficiency in 88% of cases, pathological vasoconstriction is revealed in 12%, and, in our opinion, it is one of the reasons of high arterial hypertension with development of MI in elderly patients with CKD stage 1-3. At calculation of Spearman rank correlation coefficients (Spearman coefficient) of high correlation between CAVI and coronary atherosclerosis, only IHD patients with AH without renal pathology had high correlation (Spearman coefficient 0.75). At accession and progression of renal pathology, these regularities were lost, and this coefficient in patients with unstable angina and MI on the background of CKD 1-3 stages was 0.2, i.e. our studies in patients with comorbid pathology prove that often the development of coronary atherosclerosis (at 1-2 stage of CKD, one coronary artery is more often affected, according to CAG) due to very pronounced arterial stiffness; endothelial and diastolic dysfunction with high pulmonary hypertension in comorbid patients manifests the clinic of ACS with ST-segment elevation and without ST-segment elvevation with the development of MI and UA (Ott et al. 2022).

In the analysis of CAG in IHD patients with CKD of 1-3 stages, the lesion of one coronary artery was significantly predominant; nevertheless, the development of ACS and even MI was registered quite often. In our opinion, endothelial dysfunction, arterial stiffness with high SBP, cPAP are the main factors in the development of ACS, AMI, even without hemodynamically significant stenosis in the coronary arteries, and with the progression of coronary atherosclerosis at later stages of CKD, significant differences between the groups of patients with and without kidney disease are levelled. Therefore, it is very important for such a frequent comorbid pathology of IHD, AH and CKD 1-3 stages to select etiopathogenetic treatment of AH, vascular stiffness in order to stop the process of coronary lesions at the predialysis stage of CKD. For this purpose, according to the latest Russian clinical recommendations for the treatment of AH

(Kobalava et al. 2020), we prescribed the combined hypotensive drug Amlodipine/Indapamide/Perindopril (Triplixam, Servier FC) to comorbid patients with very high values of SBP and cPAP, analysed its long-term effect on AS, PH, diastolic heart dysfunction, especially after coronary stenting and complex therapy of IHD. For this purpose, we divided IHD patients with high AH combined with CKD 1-3 stages into three groups: group 1 included patients with IHD, PICS, CHD with high AH and CKD 1-3 stages, group 2 included patients with ACS (unstable angina, MI) with high AH, CKD 1-3 stages, and group 3 included IHD, AH without renal pathology. In these three groups of comorbid patients, hypotensive combined medication including indapamide, perindopril, amlodipine was prescribed. Stenting of haemodynamically significant coronary artery stenoses was performed in 52 patients. Additionally, the patients received baseline therapy of IHD: disaggregants (cardiomagnil 75 mg/day, clopidogrel 75 mg/day or prasugrel 10 mg/day), statins (atorvastatin 40 mg/day), and B-blockers (bisoprolol 2.5-5.0 mg/day). Despite the presence of positive result after 12 weeks of therapy of IHD patients with high AH on the background of CKD 1-3 stages, we registered the degree of difference of AS, PH, diastolic heart dysfunction parameters shown in Table 2.

According to the treatment standards, all patients with IHD, AH on the background of CKD received dual antiaggregant therapy, statins, B-blockers in ACS with ST-segment elevation improvement of all indices of arterial stiffness, PH, diastolic dysfunction was faster in patients by the 10-12th day of discharge, who underwent coronary stenting according to indications, and then during 12 weeks received Amlodipine/Indapamide/Perindopril one tablet per day. Against the background of IHD baseline therapy, a 38% decrease in PWV was found, the augmentation index decreased by 20%, and on the background of coronary stenting – by 36 %.

Conclusion

1. In patients with various forms of IHD with high AH on the background of CKD of 1-3 stages maximal elevation of vascular wall stiffness indices, expressed endothelial dysfunction with predominance of insufficient vasodilation was registered in 88% of cases. Monitoring these parameters should be used to assess the effectiveness of treatment of IHD patients with CKD 1-3 stages.
2. CAG in these patients does not lead to exacerbation of CKD; three months after CAG and coronary stenting, there is a pronounced decrease in AS, SBPao, cPAP, AI with elevation of GFR, with a decrease in blood creatinine on the background of combined drug Amlodipine/Indapamide/Perindopril (Triplixam, Servier FC).
3. In patients with IHD with comorbid pathology (AH, CKD), early administration of combined three-component antihypertensive drug Amlodipine/Indapamide/Perindopril is preferable, which promotes normalisation of AS, PH, diastolic dysfunction indices with an increase in renal GFR and decrease in blood creatinine against the background of complex etiopathogenetic therapy of IHD in combination with surgical vascularisation of the heart.

Conflict of interest

The authors have declared that no competing interests exist.

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The authors have no funding to report.

Data availability

All of the data that support the findings of this study are available in the main text.

Table 2. Parameters of vascular stiffness, pulmonary hypertension, diastolic dysfunction of the heart in IHD patients with high arterial hypertension on the background of CKD 1-3 stages after coronary stenting and treatment with Amlodipine/Indapamide/Perindopril

Indices	IHD, AH on the background of CKD stage 1-3				IHD, AH without renal pathology after CS (n=42)
	Initially (n=85)	On the background of conservative therapy Amlodipine/Indapamide/ Perindopril (n=33)	ACS without ST-segment elevation, UA after CS on Amlodipine/ Indapamide/Perindopril background (n=22)	ACS with ST-segment elevation (MI) after CS on Amlodipine/ Indapamide/ Perindopril background (n=30)	
Creatinine, mmol/L	137±0.8*	118±0.7*	93±2.3*/**	95±3.4*/**	89±2.8
GFR, mmol/min/ 1.73 m^2	58±2.5*	68±3.4*	91±1.2*/**	85±2.2*/**	90±3.4
SBP, mmHg	194±3.8*	121±2.2*	118±2.1*/**	124±3.1*/**	131±3.7
DBP, mmHg	108±1.8*	92±1.4*	82±1.8*/**	101±2.8*/**	95±1.7
PWV, m/s	12.5±0.3*	8.67±0.4*	8.34±0.4*/**	8.64±0.42*/**	8.48±0.5
AI	1.59±0.2*	1.28±0.1*	1.18±0.2*/**	1.03±0.1*/**	1.09±0.1
SBPao, mmHg	169±7.2*	138±2.2*	127±3.1*/**	121±4.2*/**	115±3.2
R - CAVI	10.8±0.8*	9.0±0.4*	8.2±0.2**	8.4±0.3**	8.3±0.4
L - CAVI	10.7±0.6*	8.9±0.3*	8.3±0.1*/**	8.4±0.4*/**	8.2±0.3

Note: * – p <0.01; ** – p <0.05 – between patients on conservative therapy and coronary stenting.

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