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Research Article

The impact of fixed-dose anatihypertensive therapy on cardiorenal syndrome markers in patients with acute coronary syndrome and comorbidities

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Abstract

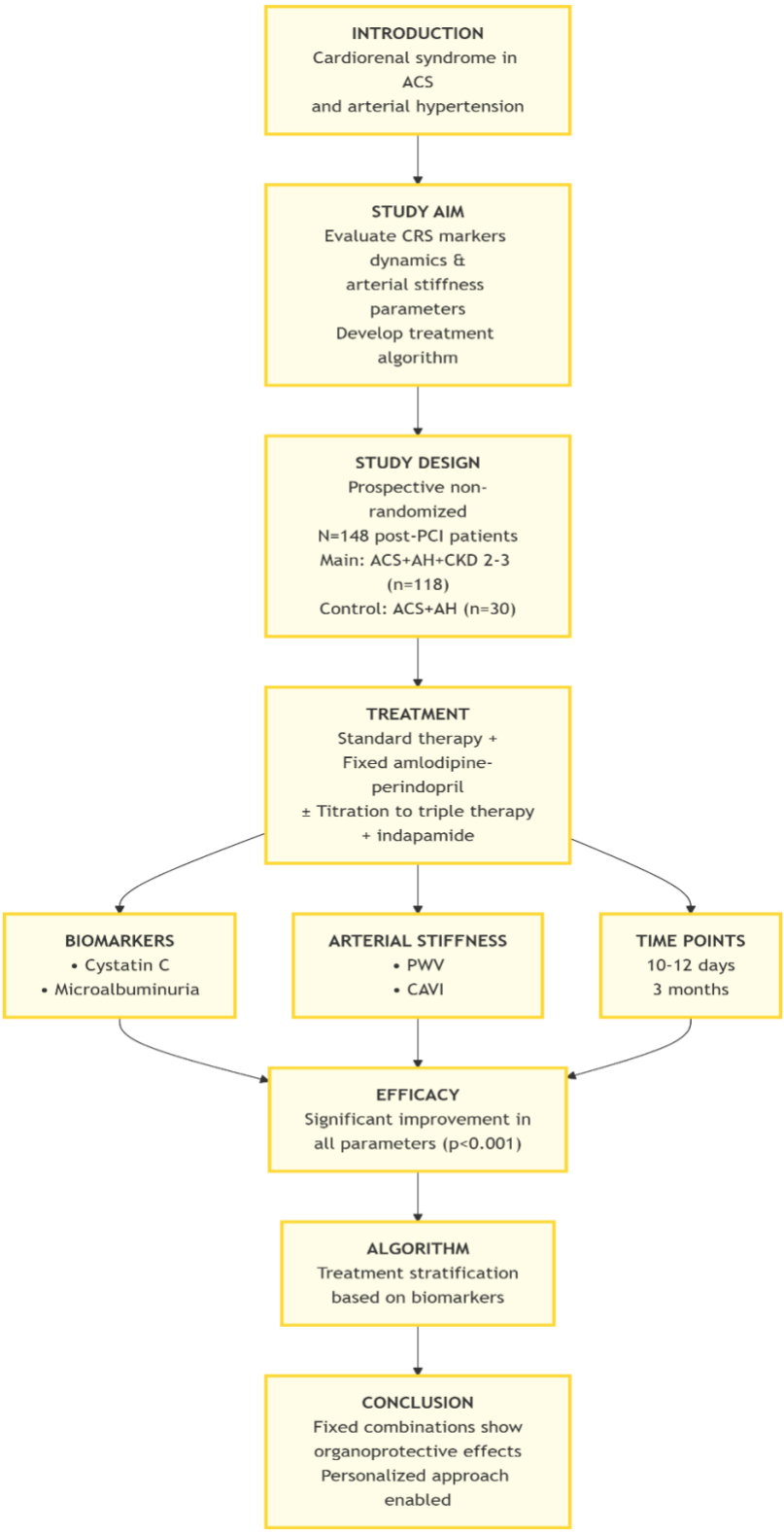
Introduction: The problem of cardiorenal syndrome (CRS) in patients with acute coronary syndrome (ACS) and arterial hypertension (AH) is highly relevant. Early diagnosis, based on cystatin C, microalbuminuria, and arterial stiffness, is key for prognosis and therapy selection. **Aim:** To assess CRS marker dynamics and arterial stiffness in patients with ACS, AH, and chronic kidney disease (CKD) on standardized therapy, and to develop a treatment selection algorithm.

Materials and Methods: A prospective study included 148 patients after percutaneous coronary intervention for ACS with AH. The main group (n=118) had CKD stages 2-3; controls (n=30) had no CKD. All received initial therapy with a fixed-dose combination of amlodipine 5 mg/perindopril 5 mg once daily, with possible uptitration to a triple combination including indapamide 1.25 mg (amlodipine 5 mg/perindopril 5 mg/indapamide 1.25 mg once daily). Markers were assessed at days 10-12 and 3 months.

Results: Cystatin C and microalbuminuria correlated with ACS severity and arterial stiffness. After 3 months, the main group showed significant improvement: cystatin C decreased from 1.35±0.33 to 1.02±0.29 mg/L; microalbuminuria – from 41.24±3.56 to 25.56±2.99 mg/L; pulse wave velocity – from 9.31 to 8.20 m/s (all $p<0.001$). Triple therapy provided a more pronounced effect. Baseline cystatin C >1.3 mg/L and microalbuminuria >30 mg/L are criteria for considering early triple therapy initiation.

Conclusion: Standardized therapy with perindopril/amlodipine, with the possible addition of indapamide, significantly improves cardiorenal status and vascular stiffness. The developed algorithm, based on initial biomarkers, allows for a personalized approach to managing these comorbid patients.

Graphical Abstract



Keywords

cardiorenal syndrome; acute coronary syndrome; chronic kidney disease; cystatin C; microalbuminuria; arterial stiffness; amlodipine; perindopril; indapamide; fixed-dose combinations; treatment algorithm; personalized medicine

Introduction

ACS, chronic forms of coronary artery disease (CAD), and post-infarction cardiosclerosis are leading causes of morbidity and mortality worldwide, including Russia. The 21st century is characterized by a rapid increase in comorbid conditions, particularly the combination of various forms of CAD with AH and CKD (Jankowski et al. 2021). The prevalence of CAD in patients with CKD is higher, and new coronary events and complications are more frequent compared to patients with CAD and preserved renal function (Virani et al. 2023). While CAD therapy is well-established, especially with modern cardiac surgical interventions, the management of patients with CAD and comorbid renal pathology is insufficiently covered in the literature, leading to diagnostic and therapeutic challenges in clinical practice (Zhivtsova et al. 2024).

Managing these patients requires not only blood pressure (BP) control but also targeted organ protection. Traditional renal function markers, such as creatinine and estimated glomerular filtration rate (eGFR), have limited sensitivity for stratifying cardiorenal risk in the acute phase of CAD. In this context, more precise biomarkers like cystatin C, which independently predicts heart failure development and mortality (Chen et al. 2022), and microalbuminuria, an indicator of generalized endothelial damage and vascular permeability (Barzilay et al. 2024), are gaining importance. Arterial stiffness serves as a crucial link between heart and kidney damage. Increased PWV and CAVI not only exacerbate hemodynamic load on the myocardium but also contribute to intraglomerular hypertension and renal fibrosis progression (Lage et al. 2022).

The search for modern diagnostic methods and the study of the effects of combined antihypertensive drugs on key pathogenetic pathways of CAD and CKD remain relevant, aiming to reduce complications and mortality from this common comorbidity, especially in emergency cardiology settings (Pribylov et al. 2022). According to the latest clinical guidelines for AH management, fixed-dose combination antihypertensive therapy should be initiated from the first step. However, clear criteria for choosing between dual and triple fixed-dose therapy in comorbid post-ACS patients, based on objective cardiorenal risk markers, are lacking.

The aim of the present study: to analyze the dynamics of CRS markers and arterial stiffness parameters in patients with ACS and AH on combined antihypertensive therapy, depending on cardiac pathology severity and CKD presence, and to develop a treatment selection algorithm based on the obtained data.

Materials and Methods

Objects under study

A prospective, controlled, non-randomized study (approved by the regional ethics committee, Minutes No. 8 dated 18.10.2022) conducted at departments of the Kursk Regional Multidisciplinary Clinical Hospital, including the regional vascular center, functional diagnostics, and ultrasound diagnostics departments. To ensure group comparability when comparing the efficacy of dual and triple therapy, analysis was performed within stratified subgroups (e.g., among STEMI patients), and statistical correction methods (multivariate regression analysis) were used to mitigate potential baseline differences.

The study included 148 patients aged 45-80 years who underwent PCI with stenting for ACS and pre-existing AH. The main group comprised 118 patients with verified CKD stages 2-3 (eGFR CKD-EPI 30-89 mL/min/1.73m²) and AH, stratified into subgroups based on ACS type: STEMI (n=45), non-ST-segment elevation myocardial infarction (NSTEMI, n=37), and unstable angina (UA, n=36). The control group consisted of 30 patients with ACS without CKD, matched by sex and age.

Exclusion Criteria: Diabetes mellitus, end-stage CKD (stages 4-5), severe comorbid pathology affecting prognosis, inflammatory kidney diseases, gout, atrial fibrillation, left ventricular ejection fraction <40%.

Research methods

Therapy protocol

All patients received standard medical therapy according to clinical guidelines, including:

- Antithrombotic therapy: Acetylsalicylic acid (Thrombo ASS, Takeda, Germany) 75 mg/day and clopidogrel (Zilt, Akrikhin, Russia) 75 mg/day;
- Hypolipidemic therapy: Atorvastatin (Atoris, KRKA, Slovenia) 20 mg/day;
- Anti-anginal and metabolic therapy: Trimetazidine (Preductal, Servier, France) 80 mg/day;
- Beta-blockers: Bisoprolol (Biprol, Zentiva, Czech Republic) 2.5-5 mg/day.

Antihypertensive therapy was conducted according to a standardized protocol based on fixed-dose combinations:

Stage I: Initial therapy – Fixed combination of amlodipine and perindopril. Starting dose: 5/5 mg once daily. If target BP was not achieved (<140/90 mm Hg for moderate risk and <130/80 mm Hg for high and very high risk), the dose was titrated to 5/10 mg, then to 10/10 mg once daily.

Stage II: If dual therapy at the maximum dose (10/10 mg) was ineffective, a switch was made to the triple fixed-dose combination – amlodipine/indapamide/perindopril at a dose of 10/2.5/10 mg once daily. The decision to switch to Triplixam was made solely based on failure to achieve target BP on the maximum dose of dual-component therapy. Cystatin C and microalbuminuria data at the visit were not considered in the clinical decision.

Assessment methods

Examination was performed on days 10-12 (Visit 1) and at 3 months (Visit 2) after PCI.

Laboratory Methods: Serum cystatin C level by immunoturbidimetric method (Lees et al. 2024), urine microalbuminuria by immunoassay, plasma endothelin-1 concentration. Lipid profile assessment (total cholesterol – TC, low-density lipoproteins – LDL, high-density lipoproteins – HDL, triglycerides – TG).

Instrumental Methods: Assessment of arterial stiffness using the VS-1500 device (Fukuda Denshi, Japan) with measurement of PWV and CAVI. Measurement of central aortic systolic pressure (cSBP), central diastolic aortic pressure (cDAP), and aortic pulse pressure. Coronary angiography (CAG) was performed in all patients upon admission for ACS.

Smoking Status Assessment: Smoking index (SI) was calculated in pack-years.

Quality of Life Assessment: Conducted using the SF-36 questionnaire and the 6-minute walk test (6MWT).

Disease Severity Assessment: Killip classification was used.

Statistical analysis

Data were processed using StatTech v.4.7.0. Quantitative data with normal distribution are described as M±SD, with non-normal distribution as Me [Q1; Q3]. For group comparisons, Student's t-test, Mann-Whitney U-test, and χ^2 test were applied. Paired tests were used to assess dynamics within groups. Correlation analysis was performed using Spearman's coefficient (ρ). Ordered logistic regression was used to assess the independent contribution of factors to disease severity. To objectively compare the efficacy of dual and triple therapy, considering the non-randomized assignment design, multivariate regression analysis was performed with adjustment for potential confounding factors (Killip class, ACS type, baseline cystatin C and microalbuminuria levels). The significance level was $p<0.05$.

Results

The groups were comparable in age, sex, and body mass index. Target BP levels were achieved in most patients in both groups (Table 1). SI was significantly higher in subgroups with more severe ACS forms (STEMI: 25.0 [12.0; 40.0] pack-years) and correlated with Killip class severity ($p=0.043$).

Table 1. Comparative characteristics of patients at baseline

Indicator	Main Group (ACS+AH+CKD, n=118)	Control Group (ACS+AH, n=30)	p-value
Age, years	64.0 [61.0; 68.0]	62.5 [58.0; 67.0]	0.215
Males, n(%)	72 (61.0)	18 (60.0)	0.922
BMI, kg/m²	28.9 [25.1; 32.5]	28.1 [24.8; 31.9]	0.481

Note: Data are presented as Me [Q1; Q3] for quantitative indicators and n (%) for categorical ones.

At study inclusion, patients with CKD showed significant differences in cystatin C and microalbuminuria levels depending on ACS severity (Table 2). The highest values were recorded in the STEMI subgroup, intermediate – in the NSTEMI subgroup, and the lowest – in the UA subgroup. Creatinine and eGFR levels did not differ significantly. Endothelin-1 levels also progressively increased from the UA group to the STEMI group. A strong direct correlation was found between cystatin C and endothelin-1 ($\rho=0.883$, $p<0.001$).

At baseline, patients with STEMI and NSTEMI and CKD had significantly higher arterial stiffness and endothelial dysfunction parameters compared to such in the control group and UA patients. Aortic pulse pressure reached maximum values in NSTEMI (62.00 [58.00; 71.00] mm

Hg) and STEMI (59.00 [50.00; 68.00] mm Hg) versus 51.50 [47.25; 54.00] mm Hg in controls (p<0.001). A similar pattern was observed for PWV – 9.35 m/s and 9.31 m/s vs. 7.47 m/s (p<0.001), and for the CAVI index (R-CAVI) – 9.20 [8.00; 10.50] and 9.10 [8.30; 10.10] vs. 8.25 [8.10; 8.78] (p<0.001), respectively.

Table 2. Comparative analysis of cardiorenal syndrome markers in main group, subgroups at visit 1

Indicator	STEMI, AH, CKD (n=45)	NSTEMI, AH, CKD (n=37)	UA, AH, CKD (n=36)	p-value
Cystatin C, mg/L, M (SD)	1.43 (0.06)*/**	1.23 (0.05)*	1.10 (0.06)	<0.001
Microalbuminuria, mg/L, M (SD)	62.07 (5.25)*/**	41.24 (3.56)*	25.56 (2.99)	<0.001
Creatinine, μmol/L, Me [IQR]	107.10 [93.60; 120.00]	111.60 [97.40; 126.90]	115.50 [97.58; 125.22]	0.664
eGFR, mL/min/1.73 m², Me [IQR]	59.91 [52.39; 73.90]	52.90 [48.89; 68.50]	60.48 [48.54; 73.94]	0.431
Endothelin-1, fmol/mL, Me [IQR]	6.40 [6.20; 6.60]*/**	5.10 [4.90; 5.40]*	4.00 [3.70; 4.20]	<0.001

Note: * – p<0.001 compared to UA, AH, CKD subgroup; ** – p<0.001 compared to NSTEMI, AH, CKD subgroup.

A weak but statistically significant direct correlation was found between cystatin C and PWV (ρ=0.205, p=0.027), and between cystatin C and R-CAVI (ρ=0.246, p=0.007).

Median TC levels were significantly higher in patients with STEMI, AH, MI, and CKD (6.24 [5.40; 7.44]) than in other groups (p < 0.001). Median TG levels also increased with worsening clinical status: from 1.35 [1.02; 1.70] in the control group (ACS with AH) to 1.92 [1.39; 2.73] in the STEMI, AH, MI, CKD group (p = 0.003). LDL levels significantly differed between groups (p < 0.001). HDL levels did not show statistically significant differences between groups (p = 0.364) (Table 3).

Table 3. Lipid profile in patients with ACS and comorbidities

Indicators	ACS, AH (n=30)	UA, AH, CKD (n=36)	NSTEMI, AH, MI, CKD (n=37)	STEMI, AH, MI, CKD (n=45)	p-value
Cholesterol, mmol/L, Me [IQR]	5.09 [4.72; 5.64]	5.76 [4.65; 6.72]	5.76 [4.92; 6.36]	6.24 [5.40; 7.44]	< 0.001*
Triglycerides, mmol/L, Me [IQR]	1.35 [1.02; 1.70]	1.44 [1.17; 2.19]	1.68 [1.38; 1.92]	1.92 [1.39; 2.73]	0.003*
LDL, mmol/L, Me [IQR]	3.25 [2.62; 3.80]	4.79 [4.05; 5.40]	4.19 [3.94; 4.73]	4.19 [3.94; 4.89]	< 0.001*
HDL, mmol/L, Me [IQR]	1.30 [1.12; 1.48]	1.30 [1.12; 1.40]	1.40 [1.20; 1.50]	1.30 [1.10; 1.50]	0.364

Note: * – statistically significant differences (p<0.05).

After 3 months of therapy, the entire main group (with CKD) showed a statistically significant positive dynamics in all key markers (Table 4), while the control group showed no changes.

Table 4. Dynamics of key indicators after 3 months of antihypertensive therapy

Indicator	Examination Period	Main Group (ACS+AH+CKD, n=118)	p-value (within group)	Control Group (ACS+AH, n=30)	p-value (within group)
Cystatin C, mg/L	Visit 1	1.35 ± 0.33	<0.001	1.32 ± 0.30	0.445
	Visit 2	1.02 ± 0.29		1.28 ± 0.28	
Microalbuminuria, mg/L	Visit 1	41.24 ± 3.56*	<0.001	39.80 ± 10.50	0.512
	Visit 2	25.56 ± 2.99*		36.50 ± 9.80	
PWV, m/s	Visit 1	9.31 [8.30-10.10]	0.003	9.15 [8.20-9.90]	0.089
	Visit 2	8.20 [7.70-9.10]		8.95 [8.10-9.70]	
CAVI	Visit 1	9.20 [8.20-10.00]	0.008	9.05 [8.10-9.80]	0.104
	Visit 2	8.40 [7.90-9.20]		8.90 [8.00-9.60]	

Note: Data are presented as M ± SD or Me [Q1; Q3]. * – averaged values for the main group; p-value calculated for comparison between Visit 1 and Visit 2 within each group.

It should be noted that all patients started therapy with a dual-component regimen. However, retrospective analysis showed that the need to switch to triple therapy was statistically significantly associated with initially higher levels of cystatin C and microalbuminuria, confirming their role as predictors of dual therapy inefficacy and the need for a more intensive approach.

To objectively assess the advantages of triple therapy over dual therapy, considering the non-randomized assignment design, regression analysis with adjustment for Killip class, ACS type, and baseline cystatin C levels was performed. After statistical correction, triple therapy remained an independent factor associated with a more pronounced reduction in cystatin C ($\beta = -0.18$, $p=0.012$) and microalbuminuria ($\beta = -0.21$, $p=0.005$) compared to dual therapy, especially in the STEMI patient subgroup.

In the control group (without CKD), it is important to note that baseline cystatin C (1.32 ± 0.30 mg/L) and microalbuminuria (39.80 ± 10.50 mg/L) levels, although statistically significantly lower than in the STEMI with CKD subgroup ($p<0.001$), were nevertheless in the borderline or slightly elevated range. This is consistent with literature data indicating that ACS itself can cause transient cystatin C elevation due to an acute-phase response and reduced renal perfusion, regardless of pre-existing CKD. However, since control group patients had no established CKD and initial kidney damage was less pronounced, marker dynamics on therapy were statistically insignificant. Thus, significant improvement in cardiorenal biomarkers was observed precisely in patients with the most pronounced initial impairments (main group with CKD), while no significant changes occurred in patients without CKD (control).

This study demonstrates that cystatin C and microalbuminuria levels are more sensitive markers of cardiorenal imbalance than traditional renal function parameters and are closely associated with the severity of an acute coronary event. The findings are consistent with large epidemiological studies confirming the role of cystatin C as an independent predictor of cardiovascular outcomes and microalbuminuria as a marker of endothelial dysfunction (Xu et al. 2021). Our study revealed no contradictions in the cardioprotective and nephroprotective effects of the studied therapy; the organoprotective effect was unidirectional.

A key result is the pronounced positive dynamics of these markers, as well as arterial stiffness parameters, specifically in patients with CKD on standardized fixed-dose combination therapy. This suggests that a structured treatment approach based on sequential use of fixed combinations of amlodipine, perindopril, and indapamide provides not only effective BP control but also significant organoprotective effects in the most severe patient category. Triple therapy demonstrated advantages in reducing arterial stiffness in patients with initially more severe vascular remodeling, consistent with data on the synergistic effects of the components on vascular function.

It is important to emphasize that although biomarker data did not influence the decision to switch to triple therapy within the protocol, their initially high levels objectively identified a group of patients requiring more intensive treatment to achieve cardiorenal protection.

Based on the study results, a therapy stratification algorithm was developed, aimed at personalizing antihypertensive treatment in comorbid patients after ACS.

1. Initiation of Basic Therapy: All patients after ACS with AH and CKD stages 2-3 are recommended to start initial therapy with a fixed combination of amlodipine and perindopril at a dose of 5/5 mg once daily.
2. Primary Examination and Risk Stratification: On days 10-12 after PCI, a comprehensive assessment of parameters is conducted: determination of the ACS clinical phenotype (STEMI, NSTEMI, UA) and laboratory determination of cardiorenal biomarker levels (serum cystatin C and urine microalbuminuria).
3. Differentiated Treatment Approach:

- For patients with STEMI and elevated biomarker levels (cystatin C > 1.3 mg/L and/or microalbuminuria > 30 mg/L), a switch to the triple fixed-dose combination of amlodipine, indapamide, and perindopril within 2 weeks is recommended. Rationale: This category is identified as having an extremely high cardiorenal risk due to combined extensive coronary damage and pronounced cardiorenal imbalance.

- For patients with STEMI and normal biomarker levels (cystatin C ≤ 1.3 mg/L and microalbuminuria ≤ 30 mg/L), a planned switch to triple therapy within 2-4 weeks is recommended. Rationale: Despite normal biomarker levels, the STEMI phenotype is associated with high long-term cardiorenal risk.

- For patients with NSTEMI/UA and elevated biomarker levels, therapy intensification by switching to the fixed triple combination within 2-4 weeks is recommended. Rationale: Elevated cardiorenal biomarker levels indicate significant cardiorenal risk even with a less severe coronary syndrome.

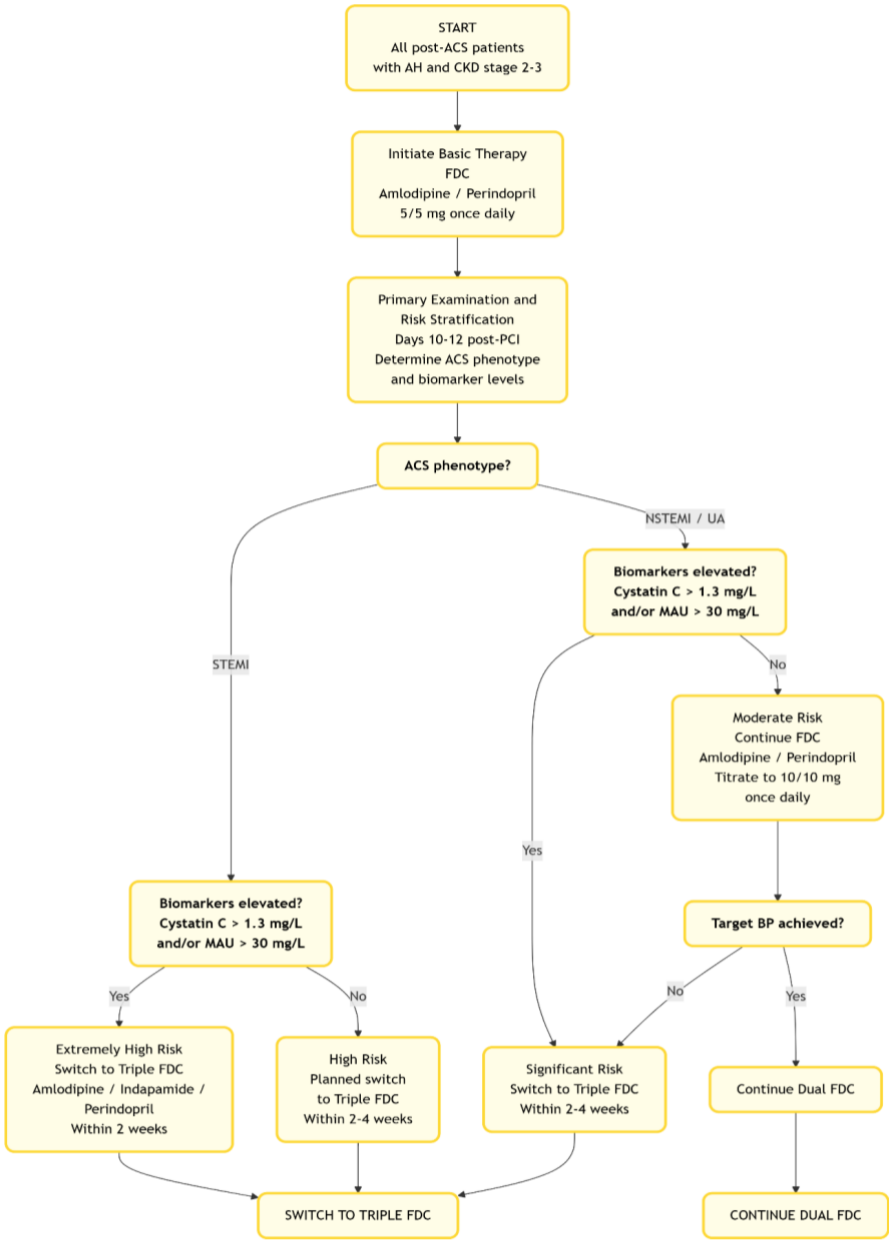


Figure 1. Algorithm for selecting antihypertensive therapy for patients after acute coronary syndrome with arterial hypertension and chronic kidney disease stages 2-3. ***Note:*** ACS – acute coronary syndrome; AH – arterial hypertension; CKD – chronic kidney disease; PCI – percutaneous coronary intervention; FDC – fixed-dose combination; MAU - microalbuminuria; BP – blood pressure; STEMI – ST-elevation myocardial infarction; NSTEMI – Non-ST-elevation myocardial infarction; UA – unstable angina.

- For patients with NSTEMI/UA and normal biomarker levels, continuation of therapy with the fixed combination of amlodipine and perindopril with dose titration to the maximum (10/10 mg once daily) to achieve target BP levels is recommended, with a subsequent switch to the fixed triple combination if target BP is not achieved. Rationale: This category has a moderate cardiorenal risk, allowing for the application of a standard antihypertensive therapy titration protocol.

The clinical significance of the proposed algorithm lies in implementing a personalized pharmacotherapy approach for comorbid patients in the post-infarction period, based on an integral assessment of two key prognostic determinants: ACS severity, determined by the clinical ACS phenotype, and the degree of cardiorenal imbalance, verified by cystatin C and microalbuminuria levels. Implementing this algorithm in clinical practice ensures the earliest possible therapy intensification in patients with the highest risk of adverse cardiorenal outcomes (STEMI combined with elevated biomarker levels), minimizes the risk of unjustified polytherapy in patients with a relatively favorable risk profile (NSTEMI/UA with normal biomarker levels),

and achieves timely organ protection through the use of fixed combinations with proven positive effects on arterial stiffness parameters and CRS markers.

Prospects for Application: The proposed algorithm requires further validation in large randomized clinical trials assessing its impact on hard endpoints.

The identified strong correlations between cystatin C, microalbuminuria, and endothelin-1 confirm the unity of pathophysiological mechanisms underlying endothelial dysfunction, kidney damage, and atherosclerosis progression. Prescribing drugs with proven vasoprotective effects, such as perindopril (ACEi) and amlodipine (CCB), as part of fixed combinations allows for comprehensive targeting of these pathogenetic pathways.

Study Limitations: The open-label, non-randomized design cannot completely exclude the influence of unaccounted factors, although the measures taken (stratification, regression analysis) minimize this possibility. The sample size and follow-up duration do not allow assessment of the impact on hard endpoints. The proposed algorithm requires validation in large randomized trials.

Conclusion

In patients with acute coronary syndrome, arterial hypertension, and chronic kidney disease, the concentrations of cystatin C and microalbuminuria are substantially elevated in more severe forms of coronary pathology, particularly STEMI, and exhibit a close correlation with markers of endothelial dysfunction, including endothelin-1, thereby serving as early indicators of cardiorenal imbalance. Individuals presenting with STEMI and comorbid conditions – specifically arterial hypertension combined with chronic kidney disease – display a more atherogenic lipid profile characterized by increased total cholesterol, triglycerides, and low-density lipoprotein cholesterol, along with a higher prevalence of multivessel coronary artery disease as evidenced by coronary angiography. The administration of standardized antihypertensive therapy based on fixed-dose combinations of amlodipine, perindopril, and indapamide is associated with significant improvement in cardiorenal syndrome markers and a reduction in arterial stiffness in this patient cohort, reflecting targeted organoprotective properties. Notably, the triple-drug regimen exerts a more pronounced beneficial effect on vascular and renal biomarkers compared with dual therapy, an advantage most clearly observed in the STEMI phenotype and validated by regression analysis adjusted for confounders. Patients with STEMI, concomitant hypertension, and stage 2–3 chronic kidney disease whose baseline cystatin C exceeds 1.3 mg/L and microalbuminuria surpasses 30 mg/L are considered candidates for an early switch to the triple fixed-dose combination of amlodipine, indapamide, and perindopril. Based on the study findings, an algorithm was developed in which these biomarkers serve as criteria for evaluating the appropriateness of such an early transition, as their elevation is associated with the need for intensified antihypertensive therapy to achieve cardiorenal protection. The data obtained regarding the efficacy of specific therapeutic regimens and the proposed biomarker-based algorithm for treatment stratification hold substantial practical value for clinical pharmacotherapy and provide a foundation for conducting large-scale randomized trials aimed at assessing their impact on long-term clinical outcomes.

Conflict of interest

Conflict of interest

The authors declare that they have no conflicts of interest.

Funding

The study received no funding.

Ethics statement

A prospective, controlled, non-randomized study (approved by the regional ethics committee, Minutes No. 8 dated 18.10.2022) conducted at departments of the Kursk Regional Multidisciplinary Clinical Hospital, including the regional vascular center, functional diagnostics, and ultrasound diagnostics departments.

Acknowledgments

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Data availability

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

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