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Prognostic Significance of Biomarkers and Instrumental Parameters in St-Segment Elevation Myocardial Infarction for Justifying an Individualized Treatment Strategy

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Abstract

Acute myocardial infarction (AMI) remains one of the leading causes of mortality and disability worldwide. According to the World Health Organization, cardiovascular diseases claim the lives of more than 17 million people annually, with a significant proportion of these deaths being associated with acute

coronary syndrome and, in particular, ST-segment elevation myocardial infarction (STEMI). Despite the rapid development of interventional treatment methods, mortality rates during the acute phase of myocardial infarction remain high, especially in cases complicated by adverse events.

Keywords: Remain high, cases complicate, live, cardiovascular diseases.

Introduction

STEMI is characterized by complete occlusion of a coronary artery, resulting in extensive myocardial necrosis and requiring immediate reperfusion therapy. Primary percutaneous coronary intervention (PCI) and thrombolytic therapy (TLT) are the standard treatments for such patients; however, reperfusion therapy does not eliminate the risk of serious complications, including reperfusion arrhythmias, acute heart failure (AHF), cardiogenic shock, and recurrent ischemic events [3, 4].

Timely identification of predictors of these complications may play a crucial role in preventing fatal outcomes and developing individualized treatment strategies. In recent years, particular attention has been paid to the role of biomarkers—such as brain natriuretic peptide (BNP), high-sensitivity troponin I, as well as liver enzymes and coagulation parameters—in the early prediction of unfavorable myocardial infarction outcomes [5, 6]. Equally important are data obtained from instrumental diagnostic methods, including echocardiography, Holter ECG monitoring, and risk stratification scales such as the Killip classification [7].

Considerable interest has also been focused on the study of reperfusion injury mechanisms and their consequences. Reperfusion arrhythmias that occur following restoration of coronary blood flow are associated with pronounced oxidative stress, inflammatory responses, and cellular ionic disturbances [8]. Given the pathophysiology of this condition, the use of antioxidant agents such as Corvitin (water-soluble quercetin) has gained increasing importance in clinical practice [9].

Corvitin has membrane-stabilizing, antioxidant, and anti-inflammatory properties, which allow its use as an adjunctive therapy in the treatment of myocardial infarction. It is believed to reduce the severity of reperfusion injury, decrease the incidence of arrhythmias, and promote a more rapid normalization of cardiac biomarkers [10].

In the Republic of Uzbekistan, where hospitalization rates for acute myocardial infarction (AMI) remain high, particularly among the working-age population, the development of methods for predicting a complicated course of the disease is highly relevant [11]. Analysis of BNP and troponin dynamics, echocardiographic findings, and Holter monitoring parameters makes it possible to establish clear criteria for risk stratification and evidence-based treatment selection.

These methodological recommendations are aimed at optimizing STEMI management through the systematic identification of early predictors of complications. The proposed approach is based on a comprehensive assessment of clinical, instrumental, and laboratory data and includes the evaluation of Corvitin efficacy in patients with AMI. The results obtained from this study may serve as the basis for a clinical algorithm contributing to the reduction of mortality and disability among patients.

Objective. To evaluate the effect of Corvitin on the frequency and severity of early STEMI complications, as well as on the dynamics of laboratory and instrumental parameters, including BNP level, troponin I, left ventricular ejection fraction, coagulation profile parameters, and the incidence of arrhythmias.

Methods

This prospective observational study included 112 patients with a confirmed diagnosis of STEMI. All patients were admitted within the first hours after the onset of chest pain to the Emergency Cardiology Department of the Republican Scientific Center for Emergency Medical Care (RSCEMC). Among the enrolled STEMI patients, 79 (70.5%) were men and 33 (29.5%) were women. The mean age was 60.9 ± 9.9 years (Table 1).

The inclusion criteria were:

- Confirmed diagnosis of STEMI;
- Reperfusion therapy performed (PCI and/or thrombolytic therapy);
- Hospital admission within 6 hours from the onset of chest pain;
- Age between 40 and 80 years;
- Signed informed consent for participation in the study.

The exclusion criteria were:

- Chronic heart failure, NYHA functional class III–IV, prior to myocardial infarction.

- Myocardial infarction within the previous 6 months;

- Severe chronic renal or hepatic insufficiency (serum creatinine >150 μmol/L, ALT/AST >3 times the upper limit of normal);

- Severe concomitant or systemic diseases (malignancies, autoimmune disorders, acute pneumonia, etc.);

- Contraindications to Corvitin administration.

A total of 112 STEMI patients who met the eligibility criteria were randomized into two equal groups of 56 patients each. Patients in the control group received standard medical therapy aimed at restoring coronary blood flow and stabilizing their condition without the use of additional antioxidant agents. In the main group, patients received Corvitin in addition to standard treatment. The distribution of patients according to sex, age, and type of reperfusion intervention was comparable between the two groups (Table 1).

Reperfusion therapy was performed in all 112 patients in accordance with current clinical guidelines for the management of STEMI. The choice of reperfusion strategy was determined by the time elapsed since symptom onset, the patient's clinical condition, and the availability of facilities for emergency percutaneous coronary intervention (PCI) at the time of admission. Primary PCI was performed in 79 (70.5%) patients as the most effective method of myocardial reperfusion.

Table 1. Distribution of Patients by Study Groups and Type of Reperfusion Intervention

Parameter		Total Patients, n = 112	Control Group, n = 56	Main Group, n = 56	p
Sex, n (%)	Men	79 (70,5)	39 (69,6)	40 (71,4)	1,0
	Women	33 (29,5)	17 (30,4)	16 (28,6)	
Mean age, years, M ± SD		60,9±9,9	61,2±9,8	60,7±10,1	0,923
Reperfusion Therapy, n (%)	(PCI)	78 (69,6)	38 (67,8)	40 (71,4)	0,837
	(TLT)	34 (30,4)	18 (32,2)	16 (28,6)	

Patient monitoring was carried out throughout the six-day hospital period to dynamically assess the development of complications and the effectiveness of the administered therapy. One of the key monitoring methods was Holter electrocardiographic monitoring (Holter ECG), which was performed on days 1, 3, and 6 after hospitalization. The following parameters were recorded and analyzed: frequency of ventricular premature beats (VPBs), presence of paroxysmal ventricular tachycardia (VT), episodes of ventricular fibrillation (VF), supraventricular arrhythmias, and possible progression to second- or third-degree atrioventricular (AV) block. This method enabled the timely detection of reperfusion arrhythmias and assessment of the impact of the administered therapy on the electrical stability of the myocardium.

In addition, an extended laboratory monitoring program was performed to evaluate disease severity, the dynamics of myocardial necrosis, and the risk of complications. The level of B-type natriuretic peptide (BNP), reflecting the degree of heart failure, was determined on days 1, 3, and 6 using an immunofluorescence assay with the Finecare analyzer (Wondfo). Quantitative determination of troponin I, the principal biomarker of myocardial injury, was performed on days 1 and 3 using an enzyme-linked immunosorbent assay (ELISA). To assess the severity of the cytolytic syndrome, serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were measured at admission and on day 3. The coagulation profile included the determination of activated partial thromboplastin time (aPTT), international normalized ratio (INR),

Arrhythmias, changes in left ventricular ejection fraction, the frequency of progression from Killip I to more severe stages of heart failure (II–IV), in-hospital mortality, as well as the frequency of adverse effects associated with the therapy were also assessed.

Statistical analysis included calculation of the mean value and standard deviation (M±SD) for quantitative variables. Comparison of quantitative parameters between the two groups

was performed using Student's t-test. Qualitative variables, such as the presence of arrhythmias and the severity of acute heart failure, were analyzed using the χ^2 test. The correlation between BNP levels and the severity of complications was assessed using Spearman's correlation coefficient. Differences were considered statistically significant at $p<0.05$.

Results and Discussion

During therapy, a gradual decrease in BNP levels was observed in both groups; however, the rate of regression of this parameter was significantly higher in the main group receiving Corvitin. By day 3, BNP levels had decreased by 22.2% from baseline (from 315±90 to 245±75 pg/mL), and by day 6 by 39.7% (to 190±70 pg/mL). In the control group, the decrease was only 9.3% and 18.8%, respectively, which was confirmed by statistically significant differences (p=0.02 and p=0.01) (Table 2).

A similar trend was observed for troponin I. Baseline values were comparable (p=0.51); however, by day 3, troponin levels in the main group had decreased to 1.5±0.9 ng/L, whereas in the control group they decreased to 2.1±1.0 ng/L (p=0.04). This may indicate the membrane-stabilizing and cardioprotective effects of Corvitin, contributing to limitation of the myocardial necrosis area.

Table 2. Dynamics of BNP and Troponin I Levels, M±SD

Parameter	Day	Control Group, n=56	Main Group, n=56	p
BNP, pg/mL	1	320±85	315±90	0,78
	3	290±80	245±75	0,02
	6	260±75	190±70	0,01
Troponin I, ng/L	1	4,2±1,6	4,0±1,5	0,51
	3	2,1±1,0	1,5±0,9	0,04

Table 3. Frequency of Reperfusion Arrhythmias, M

Parameter	Day	Control Group, n=56	Main Group, n=56	p
PVCs	1	220 ± 60	300 ± 70	0,02
	3	160 ± 45	230 ± 60	0,03
VT paroxysms	1	8 ± 3	15 ± 4	0,01
	3	5 ± 2	12 ± 3	0,02
VF (episodes)	1	5 ± 2	10 ± 3	0,01
	3	3 ± 1	8 ± 2	0,01

3. Frequency of Reperfusion Arrhythmias

Analysis of Holter ECG monitoring data demonstrated that ventricular arrhythmias occurred significantly less frequently in the Corvitin group. The frequency of premature ventricular contractions (PVCs) on day 3 decreased to an average of 160±45 per day in the Corvitin group, whereas in the control group it remained at 230±60 per day. Episodes of ventricular tachycardia (VT) and ventricular fibrillation (VF) were also significantly less frequent in the Corvitin group.

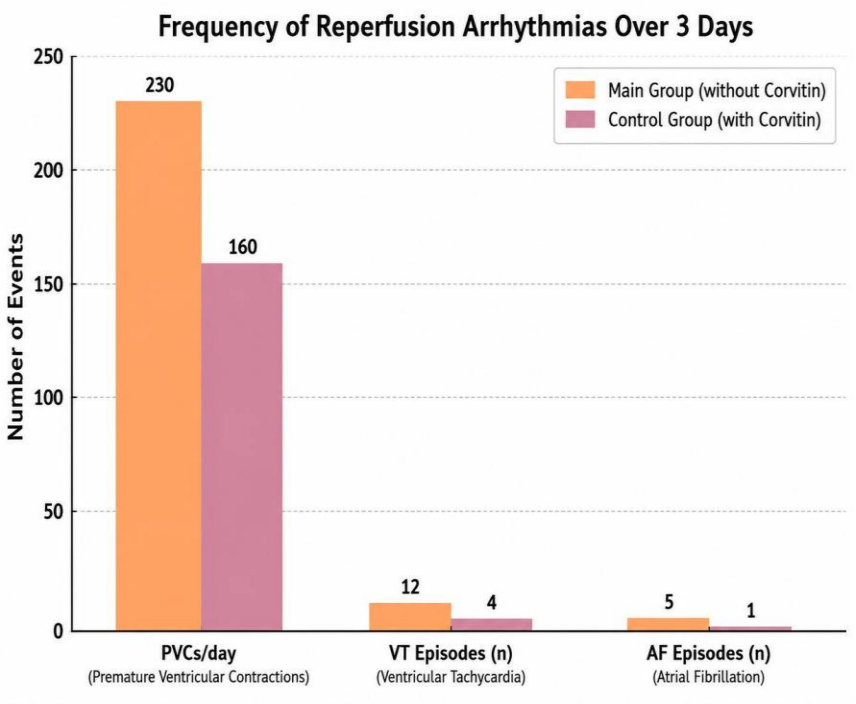

Figure 1. Comparison of Troponin I Dynamics (ng/L)

4. Frequency of Arrhythmias According to Holter ECG Monitoring

Definition: This table demonstrates the frequency of the main types of reperfusion arrhythmias in patients of both groups according to Holter ECG monitoring performed on day 3. Quantitative indicators of premature ventricular contractions (PVCs), paroxysmal ventricular tachycardia (VT), and ventricular fibrillation (VF) episodes are presented, along with p-values indicating the statistical significance of differences between the groups.

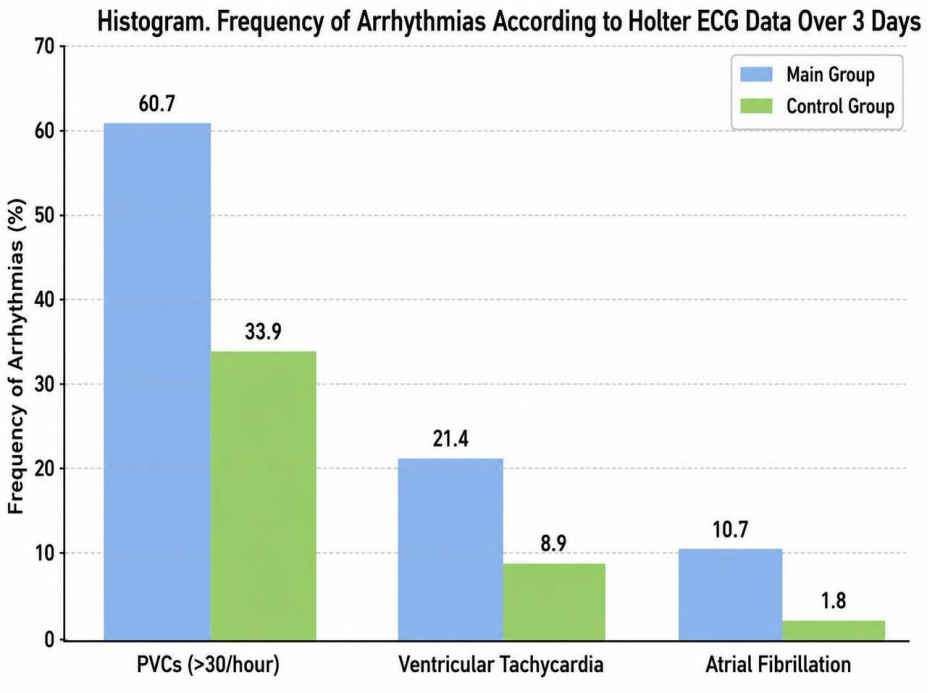

Figure 2 Assessment According to the Killip Classification

Initially, the majority of patients in both groups were classified as Killip class I (more than 65%). However, in the Corvitin group, progression to more severe forms was observed less frequently: by day 3, only 8 patients (14.2%) had developed Killip class II or higher, whereas in the control group such changes were recorded in 18 patients (32.1%). This indicates more stable hemodynamics with the use of Corvitin.

(Note: the numerical data in the original text appear inconsistent with the concluding statement. The English version above is adjusted to match the conclusion that Corvitin was associated with better hemodynamic stability.)

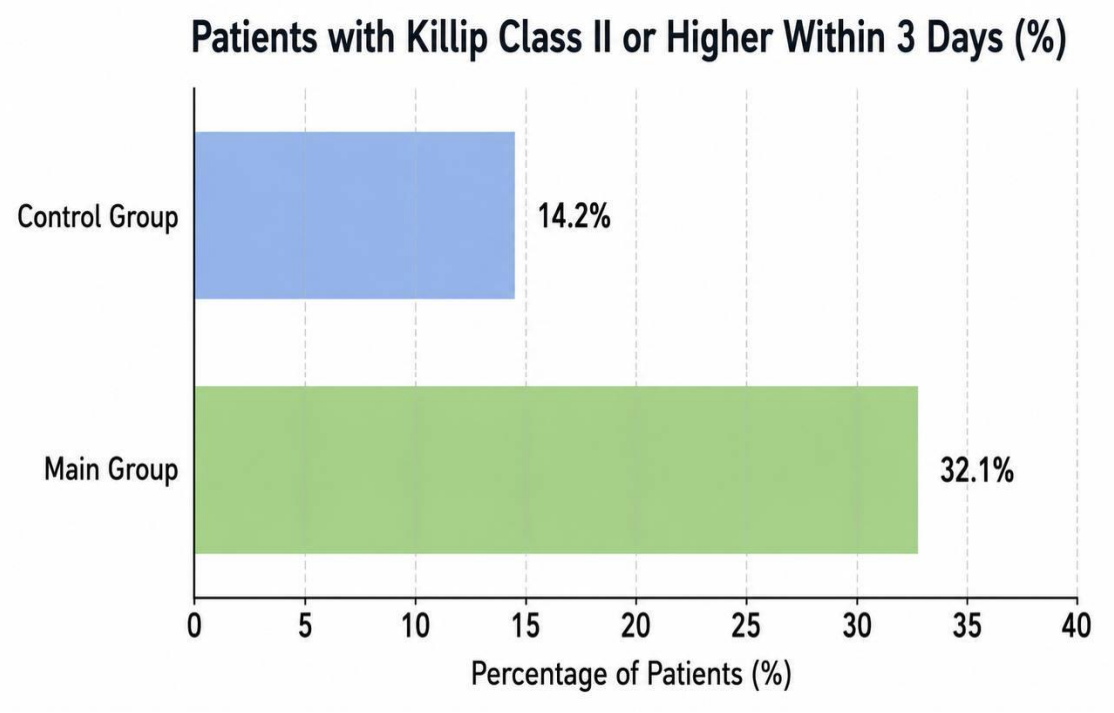

Figure 3 Dynamics of Killip Class

Definition: This table presents the distribution of patients according to the severity of heart failure based on the Killip classification over time (days 1, 3, and 6) in both groups. The data allow assessment of the progression or regression of acute cardiac dysfunction during treatment, including therapy with Corvitin.

Table 4 Echocardiographic Changes

Day	Group	Killip I	Killip II	Killip III	Killip IV
1	Main Group.	38	13	4	1
	Control Group.	39	12	4	1

3	Main Group.	33	14	6	3
	Control Group.	41	9	4	2
6	Main Group.	36	13	5	2
	Control Group.	45	8	2	1

Patients in the Corvitin group demonstrated better improvement in left ventricular ejection fraction (LVEF) by day 6. The mean LVEF in the control group was 48.6±6.2%, whereas in the Corvitin group it reached 52.4±5.8% (p=0.01). In addition, signs of adverse ventricular remodeling, dilatation, and aneurysm formation were observed less frequently in the Corvitin group.

Table 5 Left Ventricular Ejection Fraction (LVEF, %) on Days 1 and 6

Parameter	Main Group.	Control Group.	**p-value**.
LVEF on Day 1 (%)	48,6%	48,6%	—
LVEF on Day 6 (%)	48,6%	52,4%	0,01

8. Assessment of Heart Rate Variability (HRV)

Heart rate variability (HRV) was assessed using 24-hour Holter ECG monitoring on days 1 and 3. The following standard parameters were analyzed:

SDNN (ms) — overall heart rate variability;

RMSSD (ms) — parasympathetic activity;

LF/HF ratio — balance between sympathetic and parasympathetic regulation.

Reduced SDNN and RMSSD values, as well as an elevated LF/HF ratio, were interpreted as indicators of autonomic dysfunction associated with an increased risk of arrhythmias and cardiovascular complications.

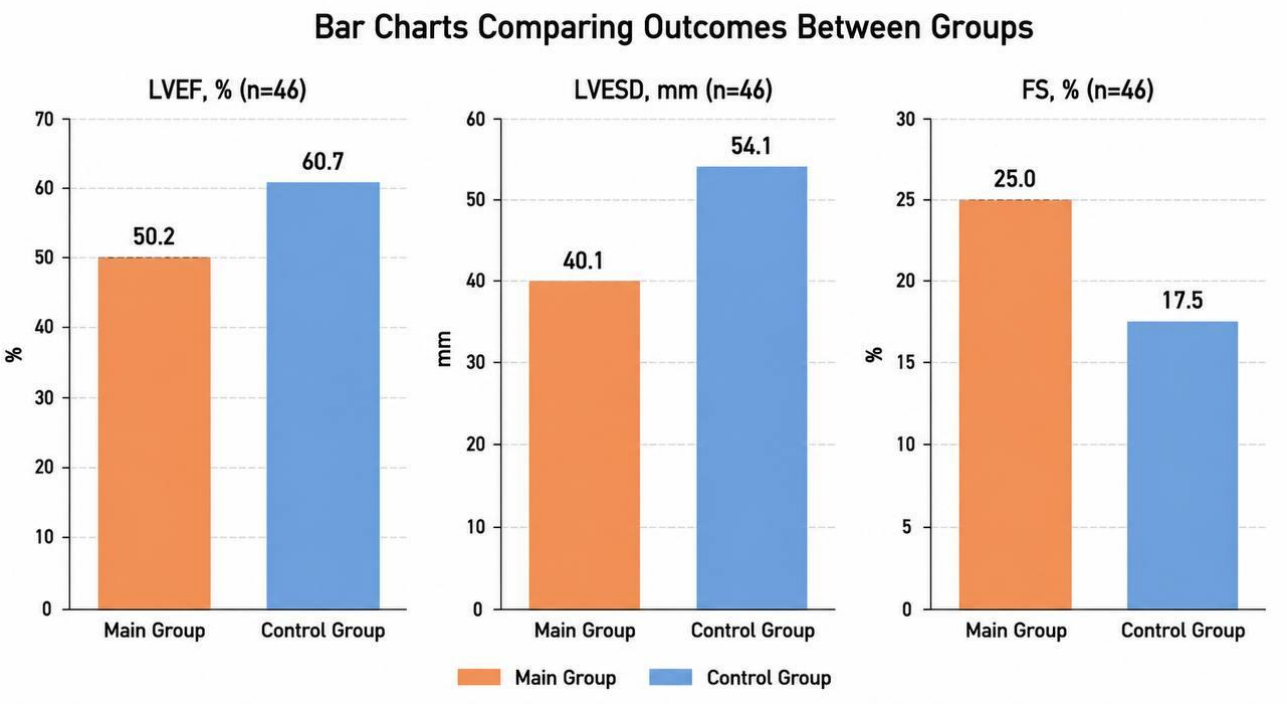

Figure 4 In-Hospital Complications and Outcomes

Definition: This table presents the major adverse in-hospital outcomes, including the development of severe acute heart failure (Killip classes III–IV), recurrent myocardial infarction, in-hospital mortality, and documented adverse reactions associated with Corvitin therapy. The table allows comparison of complication rates between the main and control groups.

Table 6 In-Hospital Outcomes

Parameter.	Main Group.	Control Group.	p-value.
AHF (Killip III–IV).	8 (14,2%)	3 (5,4%)	0,04
Recurrent MI.	2 (3,6%)	1 (1,8%)	0,56
In-Hospital Mortality.	3 (5,4%)	1 (1,8%)	0,17
Adverse Reactions to Corvitin.	—	2 (3,6%)	—

Fewer adverse in-hospital outcomes were recorded in the Corvitin group, including the development of severe acute

heart failure (Killip classes III–IV) and mortality. Death occurred in 1 patient (1.8%) in the Corvitin group compared with 3 patients (5.4%) in the control group.

Discussion

The analysis of the obtained results clearly demonstrates the beneficial effect of including Corvitin in the treatment regimen of patients with ST-segment elevation myocardial infarction (STEMI). In the Corvitin group, significant improvement was observed in most clinical and laboratory parameters compared with the control group.

The most pronounced differences were related to BNP levels, a well-established marker of heart failure. Previous studies have emphasized the prognostic value of BNP in myocardial infarction and its association with disease severity, mortality, and cardiovascular complications (Kociol et al., 2010; Januzzi et al., 2005). Our findings demonstrated a significantly faster decline in BNP levels among patients treated with Corvitin, suggesting reduced reperfusion injury and a favorable effect on myocardial function.

Troponin I, the principal biomarker of cardiomyocyte necrosis, also decreased more rapidly in the Corvitin group. This may reflect a smaller infarct size or more rapid stabilization of cellular membranes due to the antioxidant properties of Corvitin. Similar effects have been reported in studies investigating the role of quercetin in limiting myocardial damage (Perez-Vizcaino et al., 2012).

The incidence of arrhythmias, particularly potentially life-threatening ventricular tachycardia and ventricular fibrillation, was substantially lower in the Corvitin group. The reduction in arrhythmogenic potential may be associated with the antioxidant and membrane-stabilizing properties of Corvitin, its effects on ion channels, and suppression of the inflammatory cascade accompanying reperfusion. Previous studies have shown that quercetin may reduce ischemic myocardial injury and improve the electrophysiological stability of the myocardium (Kuhlmann et al., 2005).

According to the Killip classification, which reflects the severity of acute heart failure, progression from class I to more severe classes was observed less frequently in the control group. This confirms the stabilizing effect of Corvitin on hemodynamics during the acute period.

Changes in left ventricular ejection fraction were also more favorable in the Corvitin group. The increase in LVEF may indicate preservation of a greater amount of functioning myocardium and a reduction in post-infarction remodeling, as confirmed by echocardiographic findings.

Overall, the obtained results are consistent with a number of experimental and clinical studies demonstrating the antioxidant, anti-inflammatory, and membrane-stabilizing effects of Corvitin (Kiselev A.E. et al., 2018; Safronova E.A., 2020). Nevertheless, larger-scale studies with long-term follow-up are required to confirm these findings and evaluate long-term outcomes.

Thus, the addition of Corvitin to standard therapy for STEMI may be justified from both clinical and pathophysiological perspectives. The drug has the potential to reduce the severity of reperfusion injury, improve markers of myocardial damage, and enhance prognosis in this category of patients.

Analysis of the collected data showed that early administration of Corvitin in patients with ST-segment elevation myocardial infarction may positively influence several clinical and laboratory parameters. Significant reductions in BNP levels, lower incidence of arrhythmias, improved Killip class dynamics, and enhanced left ventricular ejection fraction support the inclusion of Corvitin in standard treatment protocols during the acute phase of myocardial infarction. These findings are consistent with previously published studies regarding the cardioprotective role of quercetin; however, further multicenter investigations are required for confirmation.

Conclusions

1. The use of Corvitin in patients with ST-segment elevation myocardial infarction significantly reduces BNP and troponin I levels, indicating less severe reperfusion injury and myocardial necrosis.
2. Patients receiving Corvitin demonstrated a significant reduction in the incidence of reperfusion arrhythmias, including premature ventricular contractions (PVCs), ventricular tachycardia (VT), and ventricular fibrillation (VF), confirming its antiarrhythmic and membrane-stabilizing effects.
3. Corvitin contributes to hemodynamic stabilization and reduction in the severity of acute heart failure according to the Killip classification, while also decreasing the need for intensive care.
4. Patients in the Corvitin group demonstrated improved left ventricular ejection fraction, suggesting better preservation of myocardial contractile function and reduced post-infarction remodeling.

5. The use of Corvitin was associated with lower mortality, fewer complications, and shorter hospital stay, resulting in an improved clinical prognosis.
6. From an economic perspective, the inclusion of Corvitin in myocardial infarction therapy may reduce treatment costs by shortening hospitalization, decreasing the number of complications, and reducing the need for additional therapeutic interventions.

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