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CARDIAC REMODELING IN PATIENTS UNDERGOING REGULAR HEMODIALYSIS AND THE EFFECT OF ANTIPLATELET THERAPY ON IT

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Abstract

The article presents the results of a study evaluating the six-month dynamics of cardiorenal syndrome and cardiac remodeling parameters in 101 patients with end-stage chronic kidney disease undergoing maintenance hemodialysis. It also examines the role of antiplatelet therapy in preventing cardiovascular diseases and other thromboembolic complications associated with these disorders.

Keywords: chronic kidney disease, antiplatelet agents, maintenance hemodialysis, echocardiography (EchoCG), systolic function, diastolic function, Alltrombosepin.

Introduction

In recent years, clinical studies based on echocardiographic (EchoCG) findings have demonstrated that left ventricular hypertrophy is present in more than 50% of patients with chronic kidney disease (CKD), while heart failure may remain clinically asymptomatic [7, 9]. Furthermore, left ventricular hypertrophy is detected in more than 75% of patients with end-stage renal disease.

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Multifactorial disturbances associated with renal failure, including inflammatory, neurohumoral, homeostatic, hematological, rheological, metabolic, and hemodynamic abnormalities, inevitably affect the cardiovascular system (CVS) [2, 7, 11]. Moreover, as heart failure progresses, renal function tends to deteriorate further [7, 10, 17].

When cardiac and renal dysfunction occur simultaneously in patients, the term *cardiorenal syndrome* is increasingly used in modern clinical practice [15, 20]. In most cases, chronic cardiorenal syndrome develops as a consequence of chronic heart failure (CHF) associated with various forms of ischemic heart disease (IHD) [8, 15].

Alongside several other factors, the renin–angiotensin–aldosterone system (RAAS), particularly its final component, aldosterone, plays a leading role in the development and progression of this condition [2, 4, 14].

Fibrosis is understood as an increase in the collagen fraction by two to three times above normal levels or a predominance of collagen degradation imbalance and synthesis-related processes [13, 18].

The progression of these pathological processes is particularly pronounced in patients with stage 5 chronic kidney disease (CKD) requiring extracorporeal detoxification, making this issue even more clinically relevant [12].

Therefore, we considered it necessary to conduct a scientific study aimed at assessing cardiac remodeling parameters in patients undergoing maintenance hemodialysis and evaluating the effects of antiplatelet agents on cardiac remodeling through the modulation of blood rheological properties and correction of hemostatic system disorders.

Aim of the Study

To study cardiac remodeling parameters in patients undergoing extracorporeal detoxification procedures and to evaluate the dynamics of the effect of antiplatelet therapy on these parameters.

Materials and Methods

The study included 101 patients with stage 5 chronic kidney disease (CKD) developed as a result of nephropathies of various origins and non-diabetic etiologies. The patients were under observation at the Department of Extracorporeal Detoxification of the Fergana Regional Multidisciplinary Medical Center and were receiving maintenance hemodialysis treatment at this institution.

The patients' ages ranged from 19 to 60 years, with a mean age of 39.3 ± 1.63 years. The duration of their maintenance hemodialysis treatment did not exceed 5 years and averaged 3.8 ± 1.7 years. The patients were randomly divided into Group 1 (n = 50) and Group 2 (n = 51). Only patients in Group 2 were prescribed Alltrombosepin at a dose of 200 mg/day (100 mg per capsule, twice daily).

All patients underwent echocardiographic (EchoCG) examinations at the beginning of the study and after 6 months. The examinations were conducted in the Functional Diagnostics Departments of the Fergana Regional Multidisciplinary Medical Center.

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Echocardiographic examinations were performed using a SONOSCAPE S20 ultrasound device with a 3.5 MHz transducer, employing the conventional Simpson technique.

The following parameters were determined: left ventricular end-diastolic volume (LVEDV, ml), left ventricular end-systolic volume (LVESV, ml), left ventricular end-diastolic diameter (LVEDD, mm), left ventricular end-systolic diameter (LVESD, mm), interventricular septal thickness (IVST, mm), left ventricular myocardial mass (LVMM, g), left ventricular myocardial mass index (LVMI, g/m²), stroke volume (SV, ml), and left ventricular ejection fraction (LVEF, %).

Stroke volume was calculated using the formula:

$SV = LVEDV – LVESV;$

Left ventricular ejection fraction was calculated using the formula:

$LVEF = (SV / LVEDV) \times 100\%.$

Left ventricular myocardial mass (LVMM) was calculated using the R. Devereux formula:

$LVMM = 0.8 \times \{1.04 \times [(LVEDD + PWT + IVST)^3 – LVEDD^3] + 0.6\}.$

The left ventricular myocardial mass index (LVMI, g/m²) was calculated as the ratio of LVMM to body surface area [12]. The obtained results were subjected to statistical analysis.

Results and Discussion

The following results were observed in the groups during the six-month study period.

At the beginning of treatment, LVEDV in patients of Group 1 was 151.7 ± 3.4 ml, whereas after 6 months it increased to 160.1 ± 2.64 ml. The results showed a statistically significant increase compared with the control group ($p < 0.001$), as well as a less significant increase compared with the baseline values ($p < 0.05$).

In Group 2, LVEDV at the beginning of treatment was 151.5 ± 2.94 ml, and after 6 months it increased to 154.1 ± 3.01 ml. The results showed a statistically significant increase compared with the control group ($p < 0.001$), while the change compared with the baseline values was not statistically significant.

At the beginning of treatment, LVESV in patients of Group 1 was 69.8 ± 1.69 ml, whereas after 6 months it increased to 79.4 ± 1.27 ml. The results showed a statistically significant increase compared with the control group ($p < 0.001$), as well as a statistically significant increase compared with the baseline values ($p < 0.001$).

In Group 2, LVESV at the beginning of treatment was 69.1 ± 1.65 ml, and after 6 months it increased to 73.6 ± 1.59 ml. The results showed a statistically significant increase compared with the control group ($p < 0.001$), while the change compared with the baseline values was less statistically significant ($p < 0.05$) (Table 1).

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Table 1.

Dynamics of Echocardiographic (EchoCG) Parameters in Patients Undergoing Maintenance Hemodialysis During Antiplatelet Therapy

Indicators	Control Group (n-20)	Group 1 (n-50)		Group 2 + Alltrombosepin (n-51)	
		Before Treatment	After 6 Months	Before Treatment	After 6 Months
ЧКСДХ	122,3±3,1	151,7±3,4***	160,1±2,64***^	151,5±2,94***	154,1±3,01***
ЧКССХ	47,7±1,1	69,8±1,69***	79,4±1,27***^^^	69,1±1,65***	73,6±1,59***^
ЧКССЎ	31,5±0,7	41,7±0,51***	43,6±0,48***^^	41,9±0,51***	42,7±0,53***^
ЧКСДЎ	45,7±1,2	57,8±0,65***	61,0±0,60***^^^	58,1±0,64***	58,8±0,69***
ҚАДҚ	9,5±0,9	12,4±0,30**	13,5±0,23***^^	11,9±0,33**	12,5±0,26**^
ЧҚММ	178,6±3,13	269,3±3,31***	286,2±3,30***^^^	267,6±3,27***	271,8±4,06***
ЧҚММИ	109,1±1,2	159,2±1,73***	168,7±2,03***^^^	158,9±1,67***	161,8±1,97***^
ЗҲ	74,6±1,8	81,9±1,84***	80,7±1,68**	82,4±1,79***	80,8±1,82**
ЧҚҚОФ	60,4±1,37	53,9±0,84***	49,8±0,85***^^^	54,1±0,84***	52,2±0,76***

Note: * – Differences are statistically significant compared with the control group. (*- P<0,05, **- P<0,01, ***- P<0,001); ^ - Differences are statistically significant compared with the pretreatment group. (^ - P<0,05, ^^ - P<0,01, ^^^ - P<0,001).

In echocardiography (EchoCG), among the parameters characterizing the volumetric dimensions of the heart, the left ventricular end-systolic diameter (LVESD) in Group 1 was 41.7 ± 0.51 mm at baseline and increased to 43.6 ± 0.48 mm after 6 months. This increase was statistically significant compared with the control group ($p<0.001$) and also compared with baseline values ($p<0.01$). In Group 2, the LVESD was 41.9 ± 0.51 mm at baseline and increased to 42.7 ± 0.53 mm after 6 months. The increase was statistically significant compared with the control group ($p<0.001$), while the change from baseline was less significant ($p<0.05$).

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At baseline, the left ventricular end-diastolic diameter (LVEDD) in Group 1 was 57.8 ± 0.65 mm, increasing to 61.0 ± 0.60 mm after 6 months. This increase was statistically significant compared with the control group ($p<0.001$) and compared with baseline values ($p<0.001$). In Group 2, the LVEDD was 58.1 ± 0.64 mm at baseline and increased to 58.8 ± 0.69 mm after 6 months. The difference compared with the control group was statistically significant ($p<0.001$), whereas the change from baseline was not statistically significant.

In Group 1, the interventricular septal thickness (IVST) was 12.4 ± 0.30 mm at baseline and increased to 13.5 ± 0.23 mm after 6 months. The increase was statistically significant compared with the control group ($p<0.001$) and baseline values ($p<0.01$). In Group 2, IVST was 11.9 ± 0.33 mm at baseline and increased to 12.5 ± 0.26 mm after 6 months. The change was statistically significant compared with the control group ($p<0.01$) and showed a lower level of significance compared with baseline ($p<0.05$).

Among the echocardiographic parameters reflecting myocardial mass, left ventricular mass (LVM) in Group 1 was 269.3 ± 3.31 g at baseline and increased to 286.2 ± 3.30 g after 6 months. This increase was statistically significant compared with the control group ($p<0.001$) and baseline values ($p<0.001$). In Group 2, LVM was 267.6 ± 3.27 g at baseline and increased to 271.8 ± 4.06 g after 6 months. The value was significantly higher than that in the control group ($p<0.001$), whereas the change from baseline was not statistically significant.

The left ventricular mass index (LVMI) in Group 1 was 159.2 ± 1.73 g/m² at baseline and increased markedly to 168.7 ± 2.03 g/m² after 6 months. This increase was statistically significant compared with the control group ($p<0.001$) and baseline values ($p<0.001$). In Group 2, LVMI was 158.9 ± 1.67 g/m² at baseline and reached 161.8 ± 1.97 g/m² after 6 months. The value was significantly higher than that in the control group ($p<0.001$), while the change from baseline was statistically significant at a lower level ($p<0.05$).

Among the echocardiographic parameters directly associated with cardiac function, stroke volume (SV) in Group 1 was 81.9 ± 1.84 mL at baseline and decreased to 80.7 ± 1.68 mL after 6 months. Although the value was reported as significantly different from the control group ($p<0.001$), the change from baseline was not statistically significant. In Group 2, SV was 82.4 ± 1.79 mL at baseline and decreased slightly to 80.8 ± 1.82 mL after 6 months. No statistically significant changes were observed compared with either the control group or baseline values.

The left ventricular ejection fraction (LVEF) in Group 1 was 53.9 ± 0.84% at baseline and decreased significantly to 49.8 ± 0.85% after 6 months. This decrease was statistically significant compared with the control group ($p<0.001$) and baseline values ($p<0.001$), indicating deterioration in cardiac function. In Group 2, LVEF was 54.1 ± 0.84% at baseline and decreased to 52.2 ± 0.76% after 6 months. The decrease was statistically significant compared with the control group ($p<0.001$), whereas the change from baseline was not statistically significant.

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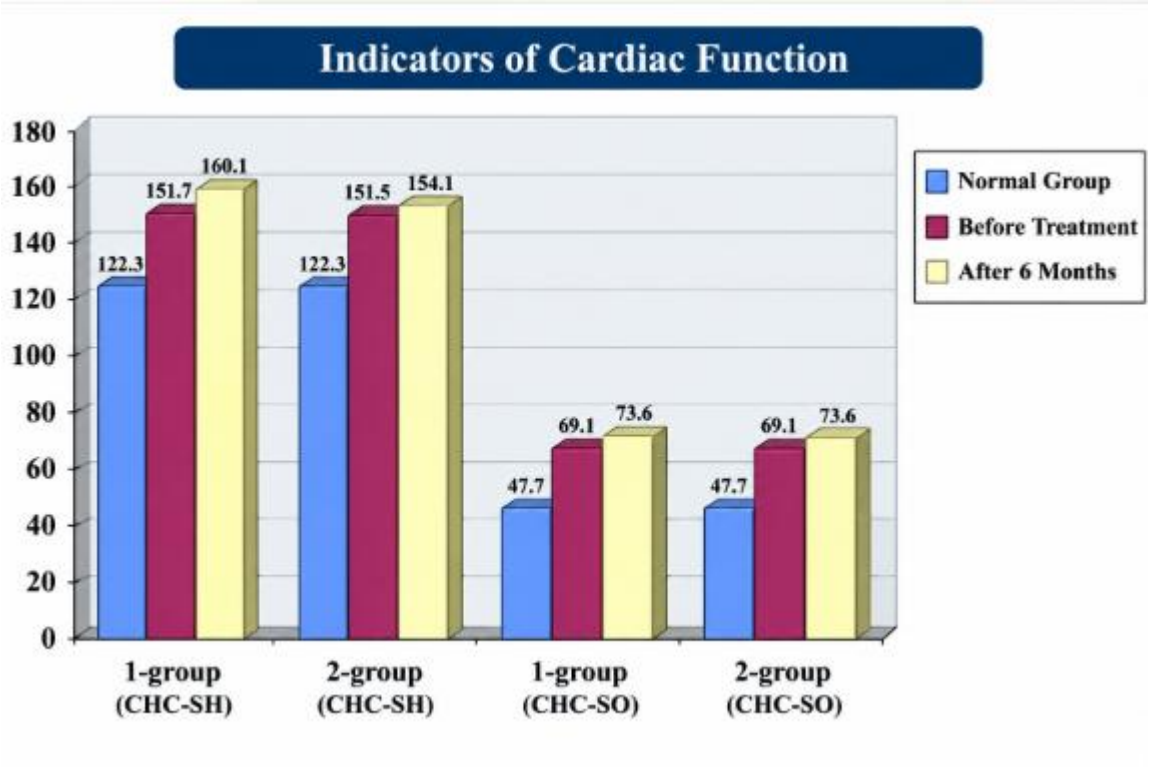


Figure 1. Comparative Dynamics of Left Ventricular End-Diastolic Volume (LVEDV) and Left Ventricular End-Systolic Volume (LVESV) During Antiplatelet Therapy

Analysis of the diagram revealed that left ventricular end-diastolic volume (LVEDV) and left ventricular end-systolic volume (LVESV) were significantly higher in both patient groups than in the control group, both at baseline and after six months of treatment ($p < 0.001$). However, compared with baseline values, patients in Group 1 demonstrated further increases in these parameters after six months, with varying degrees of statistical significance (up to $p < 0.001$), indicating the continued progression of adverse cardiac remodeling. In contrast, patients in Group 2 who received Alltrombosepin exhibited no statistically significant changes in these parameters compared with their baseline values. These findings suggest that Alltrombosepin may have attenuated the progression of adverse cardiac remodeling, potentially through its antiplatelet effects (Figure 1).

The echocardiographic parameters reflecting cardiac function demonstrated the following changes. According to the results, stroke volume (SV) was significantly higher in both patient groups compared with the control group ($p < 0.001$). However, no statistically significant changes in SV were observed in either group during the follow-up period compared with baseline values. Left ventricular ejection fraction (LVEF) was significantly

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lower in both patient groups than in the control group ($p < 0.001$). Furthermore, patients in Group 1 demonstrated a statistically significant reduction in LVEF after six months compared with baseline ($p < 0.001$), indicating a deterioration in left ventricular systolic function. In contrast, patients in Group 2 receiving the antiplatelet agent Alltrombosepin exhibited only a statistically nonsignificant decrease in LVEF compared with baseline values, suggesting a relatively stable course of left ventricular systolic function during the observation period (Figure 2).

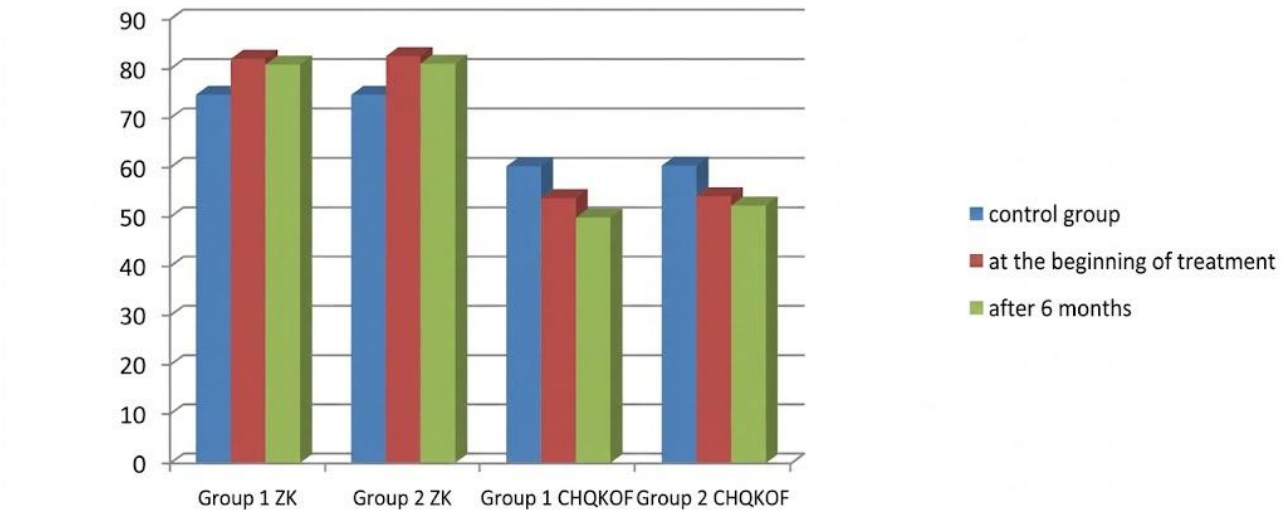


Figure 2. Comparative Dynamics of SV and EF Parameters During Antiplatelet Therapy.

Thus, in patients receiving maintenance hemodialysis, inhibition of platelet aggregation occurs from the first days of Alltrombosepin administration. This contributes to improvements in blood rheological properties and the correction of hemostatic disorders, as well as helping to prevent thromboembolic complications. Consequently, it may slow the progression of chronic cardiorenal syndrome and relatively reduce the risk of cardiovascular mortality.

Furthermore, progressive deterioration of cardiac function activates the sympathoadrenal system, which subsequently leads to activation of the renin–angiotensin–aldosterone system [1, 6, 21]. As a result, sodium and water retention increases, accompanied by glomerular hypertension and glomerular injury. These processes reduce the effectiveness of detoxification therapy, while fluid retention increases central venous pressure and consequently increases the preload on the left-sided cardiac chambers [3, 5]. At the same time, the accumulation of angiotensin II and its interaction with angiotensin receptors lead to vasoconstriction and arteriolar hypertrophy, thereby increasing left ventricular afterload [16, 19, 22].

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Thus, the administration of antiplatelet therapy in patients receiving maintenance hemodialysis may partially interrupt this pathological cascade, mitigate the consequences of cardiorenal syndrome, and potentially reduce the risk of mortality associated with this condition.

Conclusion

Thus, cardiac remodeling occurs during hemodialysis as a result of pronounced hemodynamic changes and the development of cardiorenal syndrome. The use of antiplatelet agents is important in patients receiving maintenance hemodialysis to control cardiorenal syndrome, slow the progression of cardiac remodeling, and prevent cardiovascular complications.

Regular administration of antiplatelet agents in patients undergoing maintenance hemodialysis helps prevent excessive activation of the blood coagulation cascade. Consequently, this may contribute to the management of cardiorenal syndrome and the slowing of cardiac remodeling.

The regular use of Alltrombosepin as an antiplatelet agent in patients receiving maintenance hemodialysis may help prevent the progression of cardiac remodeling and, consequently, potentially reduce the risk of mortality associated with cardiovascular failure.

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