International Journal of Medical Sciences And Clinical Research

VOLUME Vol.06 Issue01 2026
PAGE NO. 21-23
DOI 10.37547/ijmscr/Volume06Issue01-06

Effect of Cardiac Glycosides on Myocardial Hibernation in Ischemic Heart Disease

Abdullaeva Ch.A.
Tashkent State Medical University, Uzbekistan

Irisov J.B.
Tashkent State Medical University, Uzbekistan

Zaxidova M.U..
Tashkent State Medical University, Uzbekistan

Juraev M.I.
Tashkent State Medical University, Uzbekistan

Mullaeva S.I.
Tashkent State Medical University, Uzbekistan

Received: 12 December 2025; **Accepted:** 30 December 2025; **Published:** 12 January 2026

Abstract: A total of 30 patients with ischemic heart disease complicated by chronic heart failure (CHF) were enrolled in the study. In nearly all patients, CHF of a moderate functional class according to the NYHA classification was observed. At rest, echocardiographic examination assessed the following main parameters: left ventricular end-systolic volume (LVESV), left ventricular end-diastolic volume (LVEDV), stroke volume (SV), and left ventricular ejection fraction (EF).

In patients with ischemic heart disease complicated by CHF, inotropic stimulation of the left ventricle by cardiac glycosides leads to a significant reduction in end-diastolic volume and dimensions. An overall and segmental increase in ejection fraction indicates the reversibility of left ventricular myocardial reserve function.

Keywords: Chronic heart failure, inotropic stimulation, cardiac glycosides.

Introduction:

Ischemic heart disease (IHD) is one of the most common diseases and one of the leading causes of mortality, as well as temporary and permanent disability of the population, both worldwide and in Uzbekistan [2,4,6,7,8]. The wide prevalence of IHD and the presence of a large number of complications, especially heart failure (HF), which plays an undeniable role in determining the prognosis of the disease, underline the relevance of this problem. At present, the diagnosis of HF is extremely difficult due to the absence of subjective symptoms of the disease and, as a rule, late or incidental visits to physicians [1,2,3,7,8].

The neurohumoral system becomes involved in the pathological process already at the early stages of chronic heart failure (CHF). On the one hand, its activation contributes to the compensation of cardiac function in response to a decrease in cardiac output; on the other hand, it stimulates the progression of decompensation and the development of irreversible changes in the body. Activation of the neurohumoral system is the most important link in the pathogenesis of CHF, a marker of the presence and progression of the disease, and a target for therapeutic interventions [5,8,9,11,17].

Determination of the concentrations of a number of

neurohormones involved in the pathogenesis of the disease makes it possible not only to assess the severity of CHF and predict patient outcomes, but also to identify specific targets that should be addressed in the treatment of CHF [1,9,13,16].

Currently, the leading role of the sympathoadrenal system (SAS) in the development and progression of CHF is widely recognized. Correction of increased SAS activity with β-blockers is accompanied by improvement in both the clinical condition and prognosis of patients with CHF. Modern principles of HF therapy require new approaches that influence the processes of cardiovascular remodeling and the mandatory combination of drugs with different pharmacodynamic properties, taking into account the complex and multifactorial genesis of the disease [2,10,17,16].

Objective of the study: To identify the effect of glycosides on hibernating myocardium by comparing hemodynamic parameters in patients receiving glycosides.

Materials and Methods

The study included 30 patients with ischemic heart disease (IHD) complicated by chronic heart failure (CHF) who were admitted to the cardiology department of the Republican Scientific Center for Emergency Medical Care. The mean age of the patients was 66.7 ± 10.8 years; of the 30 patients, 18 (60%) were men and 12 (40%) were women. The diagnosis of exertional angina, functional class (FC) III, was established in accordance with the criteria of the Canadian Cardiovascular Society (1976), as proposed by the WHO Expert Committee of the All-Union Cardiology Research Center of the USSR Academy of Medical Sciences (1984).

Almost all patients had chronic heart failure of moderate functional classes according to the NYHA classification. The control group consisted of 16 practically healthy individuals matched by age and sex.

Therapy for IHD included a hypolipidemic diet; antiplatelet agents (aspirin, 125 mg/day); beta-blockers (bisoprolol, 2.5–5 mg once daily); statins (simvastatin, 10 mg once daily); nitrates (Cardiket, 20 mg twice daily); angiotensin-converting enzyme (ACE) inhibitors as indicated (enalapril, 2.5–10 mg twice daily); and diuretics (furosemide, 20–40 mg/day; spironolactone, 25–50 mg/day). In addition to standard therapy, patients in the main group received digoxin (0.25 mg once daily) as a cardiac glycoside.

On days 8–10 and after 3.5 ± 1.2 months, all patients underwent echocardiographic (EchoCG) examination using a Siemens Sonoline Omnia ultrasound system (Germany) with a multifrequency 2–4 MHz transducer. Imaging was performed using standard views: parasternal long- and short-axis views; short-axis views at three levels (mitral valve, papillary muscles, and apical level); and apical two- and four-chamber views.

At rest, the following main echocardiographic parameters were assessed: left ventricular (LV) end-systolic volume (ESV), LV end-diastolic volume (EDV), stroke volume (SV), and LV ejection fraction (EF). ESV and EDV were calculated using the area–length method modified by Simpson (disk method) from the apical view. EF was calculated using the formula:

EF = (EDV − ESV) / EDV,

and indexed values of LV end-systolic and end-diastolic volumes were also evaluated.

Results and Discussion

A comparative analysis of selected clinical and instrumental parameters of patients in both groups is presented in Table 1. No significant differences between the groups were found in terms of sex and age distribution, presence of atrial fibrillation, history of myocardial infarction, IHD, or clinical manifestations of heart failure.

According to resting echocardiographic data obtained on day 1 of the disease, moderate dilation of the LV cavity was observed: LV end-systolic dimension (ESD) 4.5 ± 0.1 cm, LV end-diastolic dimension (EDD) 6.0 ± 0.1 cm, LV ESV 95.5 ± 4.1 ml, and LV EDV 168.5 ± 5.8 ml. At that time, reduced myocardial contractility was associated with cardiac rhythm disturbances, zones of necrosis (i.e., irreversible myocardial dysfunction), as well as coexisting zones of reversible myocardial dysfunction (stunned and hibernating myocardium).

On days 7–10, prior to stress echocardiography, resting EchoCG showed persistently reduced systolic function; however, a significant increase in fractional shortening and overall EF was noted, reaching 25.5 ± 0.6% and 46.4 ± 0.6%, respectively (compared with 22.5 ± 0.6% and 43.1 ± 0.9% on day 1). LV ESD was 4.35 ± 0.2 cm and LV EDD was 5.8 ± 0.1 cm. A significant reduction in LV ESV by 15% and LV EDV by 11.2% was also observed. Segmental LV ejection fraction on days 7–10 increased insignificantly compared with baseline values.

Conclusions

1. In patients with IHD, reduced indices of LV systolic function may be caused not only by irreversible myocardial changes but also by concomitant reversible myocardial alterations. Inotropic stimulation of the LV with cardiac glycosides in patients with IHD complicated by CHF leads to a significant reduction in end-systolic dimensions and volume, as well as an increase in global and segmental EF, indicating

reversible LV dysfunction with preserved myocardial reserve.

2. The use of dobutamine stress echocardiography in patients with IHD and LV dysfunction allows an objective assessment of global and regional myocardial systolic function and enables clear identification of zones of reversible (hibernation, stunning) and irreversible myocardial dysfunction.

References

1. Amosova E.N. (2001) Sovremennye metody lecheniya bol'nykh, perenesshikh infarkt miokarda, i vtorichnaya profilaktika obostreniya ishemicheskoi bolezni serdtsa. Ukr. kardiol. zhurn., 4: 97–104.
2. Belenkov Yu.N., Ageev F.T., Mareev V.Yu. Znakom'tes': diastolicheskaya serdechnaya nedostatochnost'. Serd. nedost., 2000; 2: 58–61.
3. Belenkov Yu.N. Rol' narushenii sistoly i diastoly v razvitii serdechnoi nedostatochnosti. Ter. arkhiv, 2000; 9: 3–7.
4. Dolzhenko M.M., Potapov S.V., Fedotov S.V. (2001) Postinfarktnaya ishemiya miokarda: prospektivnye dostizheniya remodelirovaniya levogo zheludochka. Ukr. kardiol. zhurn., 5: 28–33.
5. Dyadyk A.I., Bagrii A.E. Vliyanie razlichnykh doz Veroshpirona na postinfarktnoe remodelirovanie levogo zheludochka. Ukr. kardiol. zhurn., 2003, №2 (34) – III/IV: 79–83.
6. Dyadyk A.I., Bagrii A.E. Sovremennye podkhody k lecheniyu patsientov, perenesshikh infarkt miokarda. Ukr. kardiol. zhurn., 2002, №1: 19–31.
7. Martynov A.I., Vasyuk Yu.A., Kopeleva N.V., Krikulov P.V. Postinfarktnoe remodelirovanie levogo zheludochka: vozmozhnosti beta-blokatorov. Kardiologiya, 2002, №3: 61–64.
8. Ol'binskaya L.I., Sizova Zh.M. Khronicheskaya serdechnaya nedostatochnost'. Monografiya. Moskva: «Reafarm», 2001; 344 s.
9. Parkhomenko A.N., Irkin O.I. Postinfarktnoe remodelirovanie serdtsa: patogenez i podkhody k optimizatsii terapii. Ukr. kardiol. zhurn., 2002, №6: 29–38.
10. Sledzevskaya I.K., Babii L.N., Savitskii S.Yu., Stroganova N.P., Fisenko L.I. Remodelirovanie levogo zheludochka i prognoz techeniya ishemicheskoi bolezni serdtsa u bol'nykh, perenesshikh infarkt miokarda v zavisimosti ot nalichiya arterial'noi gipertenzii. Ukr. kardiol. zhurn., 2002, №2: 14–18.
11. Tarasov N.I., Sizova I.N., Malakhovich E.V., Barbarash O.L. Znachenie pokazatelei vnutriserdechnoi i legochnoi gemodinamiki v razlichnye periody infarkta miokarda dlya rannei diagnostiki postinfarktnogo remodelirovaniya serdtsa. Ros. kardiol. zhurn., 2001, №1: 20–22.
12. 憬 Florya V.G. (1997) Rol' remodelirovaniya levogo zheludochka v patogeneze khronicheskoi nedostatochnosti krovoobrashcheniya. Kardiologiya, 5: 63–69.
13. 憬 Abbate A., Biondi-Zoccai G.G.L., Bussani R. et al. (2003) Increased myocardial apoptosis in patients with unfavorable left ventricular remodeling and early symptomatic post-infarction heart failure. J. Am. Coll. Cardiol., 41: 753–760.
14. Banerjee P, Banerjee T, Khand A, et al. Diastolic heart failure; neglected or misdiagnosed& JASS 2002; 39; 138-141.
15. Braunwald E., Antman E.M., Beasley J.W. et al. (2002) ACC/ AHA 2002 Guideline update for the management of patients with unstable angina and non-ST-segment elevation Myocardial Infarction. J. Am. Coll. Cardiol., 40: 1366–1374.
16. Cicoira M., Zanolla L., Rossi A. et al. (2002) Long-term, dose-dependent effects of spironolactone on left ventricular function and exercise tolerance in patients with chronic heart failure. J. Am. Coll. Cardiol., 40: 304–310.
17. Chatterjee K. Primary diastolic heart failure. Am J Geriatr Cardiol 2002; 11; 178- 187.
18. Cohn JN, Ferrari R, Sharpe N. Cardiac remodeling – concepts and clinical implications; a consensus paper from an international forum on cardiac remodeling/ JACC 2000;35;569-582.
19. Farquharson C.A.J., Struthers A.D. (2000) Spironolactone increases nitric oxide bioavailability, improves endothelial vasodilator dysfunction, and suppresses vascular angiotensin I/angiotensin II conversion in patients with chronic heart failure. Circulation, 101: 594–597.
20. Hunt S.A., Baker D.W., Chin M.H. et al. (2001) ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to revise the 1995 Guidelines for the Evaluation and Management of Heart Failure). J. Am. Coll. Cardiol., 38: 2101–2113.