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# Characteristics and prognostic assessment of acute myocarditis in patients under martial law in Ukraine: long-term follow-up results

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**The aim of the study** – to investigate the course of myocarditis in patients, including combatants, with different risk profiles and to determine prognostic factors for an unfavorable outcome over 24 months of observation under martial law conditions.

**Material and methods.** A total of 204 patients with AM (134 men and 70 women; mean age 38.2±8.6 years), including 90 combatants, were examined. Patients were stratified into three risk categories: the 1<sup>st</sup> group consisted of 80 high risk patients with de novo reduced LVEF ( $\leq 40\%$ ), clinical signs of heart failure, and extensive LGE on cardiac MRI; the 2<sup>nd</sup> group comprised 64 intermediate risk patients with moderately reduced LVEF (41–49%) and  $\geq 2$  affected LV segments on MRI; the 3<sup>rd</sup> group included 60 low risk patients with preserved LVEF ( $\geq 50\%$ ) and  $< 2$  affected LV segments. All patients underwent speckle-tracking echocardiography, 24-hour ECG monitoring with arrhythmia and HRV assessment, cardiac MRI with LGE. Comprehensive diagnostic assessments were performed within the AM onset and repeated at 12 and 24 months.

**Results and discussion.** Predictors of persistent myocarditis over the 24-month follow-up were identified: baseline LV EDVi  $\geq 105$  mL/ml, LVEF  $\leq 40\%$ , GLS  $\leq 9.0\%$ , GCS  $\leq 8.0\%$ , presence of NSVT paroxysms, a total of  $\geq 5$  affected LV segments and  $\geq 3$  LV segments with inflammatory involvement. Predictors of transition to dilated cardiomyopathy included an LV EDVi  $\geq 105$  mL/ml, LVEF  $< 30\%$ , GLS  $\leq 8.0\%$ , GCS  $\leq 7.0\%$ , a total of  $\geq 9$  affected LV segments, and  $\geq 5$  LGE-positive segments during baseline investigation. Predictors of recovery at 24 months were an LV EDVi  $\leq 85$  mL/ml, LVEF  $\geq 50\%$ , GLS  $\geq 11.0\%$ , GCS  $\geq 12.0\%$ , involvement of  $\leq 3$  LV segments, and absence of NSVT during the baseline assessment within the first month after symptom onset. Among combatants with myocarditis predictors of persistent frequent supraventricular ectopy and atrial fibrillation at 24 months included a HADS anxiety score  $\geq 11$ , HAM-A score  $\geq 25$ , LF/HF ratio  $\leq 1.20$ ,  $\geq 5$  affected LV segments, and  $\geq 2$  LV segments with LGE on baseline MRI. Additional predictors of persistent atrial fibrillation included an LV EDVi  $\geq 105$  mL/ml and LVEF  $\leq 40\%$ .

**Conclusions.** Results of the study refined contemporary approaches to prognosticating the clinical course of myocarditis and identified predictors of recovery, long-term disease persistence and transition to dilated cardiomyopathy. Among combatants with myocarditis predictors of persistent frequent supraventricular ectopy and atrial fibrillation paroxysms at 24 months were established, along with an association between these arrhythmias, the presence of anxiety and impaired heart rate variability.

**Key words:** myocarditis, left ventricle dysfunction, arrhythmias, imaging, heart rate variability, prognosis, dilated cardiomyopathy, combatants

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**M**ycocarditis is characterized by an inflammatory involvement of the myocardium, which clinically may be expressed through a broad and heterogeneous spectrum of symptoms, ranging from asymptomatic forms to fulminant heart failure (HF) [1]. Despite advances in cardiovascular imaging, the evolution of magnetic resonance techniques, and progress in pharmacotherapy, myocarditis remains a diagnostic and therapeutic challenge, limiting the ability to accurately predict its early course. Acute myocarditis (AM) prognosis is largely determined by the degree of left ventricular (LV) dysfunction at the onset, the response to medical therapy, and subsequent changes in clinical and functional parameters [2, 3]. Evidences from long-term follow-up in patients with AM remains limited and is derived predominantly from observational studies [4]. This determines the need to search for new approaches to risk stratification and personalized surveillance strategy development. Early identification of patients at increased risk for persistent arrhythmias or chronic inflammatory activity, LV dysfunction is crucial for optimizing treatment and preventing adverse outcomes. Early identification of such high-risk patients makes it possible to optimize targeted therapeutic decisions, reduces the frequency of hospitalizations, and improves long-term prognosis. Special attention should be given to combatants in whom chronic stress, excessive physical exertion and sustained psychoemotional overload may trigger AM or exacerbate its clinical course. In the context of the increasing prevalence of stress-associated cardiovascular disorders the problem of timely diagnosis and identification of early prognostic markers of complicated AM is of particular medical and social importance.

**The aim** – to investigate the course of myocarditis in patients, including combatants, with different risk profiles and to determine prognostic factors for an unfavorable outcome over 24 months of observation under martial law conditions.

## MATERIAL AND METHODS

In total 204 patients (134 men and 70 women; mean age  $38.2 \pm 8.6$  years) including 90 active combatants (68 men and 22 women; mean age  $39.1 \pm 8.0$  years) were hospitalized due to AM in the Institute's clinic during the period of martial law in Ukraine from 2022 through August 2025. Before enrolment all participants were fully informed about the diagnostic procedures and treatment performed and provided written informed consent for the processing of personal data. The diagnosis of AM was based on clinical presentation, laboratory findings, electrocardiographic (ECG)

and echocardiographic (EchoCG) assessment and cardiac magnetic resonance imaging (MRI) with late gadolinium enhancement (LGE) in accordance with the updated Lake Louise criteria [5–8]. Patients were stratified into 3 groups according to the risk assessment: the 1<sup>st</sup> group included 80 high risk patients who presented with de novo reduced left ventricular ejection fraction (LVEF  $\leq 40\%$ ), clinical signs of HF and extensive LGE on cardiac MRI; the 2<sup>nd</sup> group comprised 64 intermediate risk patients with moderately reduced LVEF (41–49%) and involvement of  $\geq 2$  LV segments; the 3<sup>rd</sup> group consisted of 60 low risk patients with preserved LVEF ( $\geq 50\%$ ) and involvement of  $< 2$  LV segments [1].

During 24-hour ECG monitoring the total number of supraventricular ectopic beats (SVEB) and ventricular ectopic beats (VEB) per day as well as atrial fibrillation (AF) and non-sustained ventricular tachycardia (NSVT) paroxysms were assessed. Frequent VEB and SVEB were defined as a burden of  $\geq 1.0\%$  relative to the total number of normal sinus beats recorded over 24 hours. Heart rate variability (HRV) was assessed using the frequency parameter SDNN (standard deviation of all RR intervals) and spectral parameters: the low frequency (LF, 0.04–0.15 Hz) and high-frequency (HF, 0.15–0.5 Hz) spectral components; sympathovagal balance was quantified by the LF/HF ratio [9]. By transthoracic EchoCG LVEF was assessed using the biplane Simpson's method and end-diastolic volume was indexed to body surface area to obtain the end-diastolic volume index (EDVi) [10]. Global longitudinal strain (GLS) and global circumferential strain (GCS) were measured by speckle-tracking EchoCG [11]. HF functional class (FC) was determined according to the New York Heart Association (NYHA) criteria based on the 6-minute walk test (6MWT). A walking distance of 426–550 m corresponded to NYHA FC I, 300–425 m – to FC II, 150–300 m – to FC III, and  $< 150$  m – to FC IV.

Anxiety was assessed using the Hospital Anxiety and Depression Scale for Anxiety (HADS-A) which contains 7 items with a 4-point gradation (0–3) and allows evaluating symptoms from 0 to 21 points. Scores  $\geq 11$  were interpreted as indicative of clinically significant anxiety [12]. Anxiety severity was further evaluated using the Hamilton Anxiety Rating Scale (HAM-A), where  $\leq 17$  points corresponded to the absence/mild anxiety, 18–24 points indicated moderate anxiety and  $\geq 25$  points – severe anxiety.

Patients received guideline-directed medical therapy for HF, including angiotensin-converting enzyme inhibitors or sacubitril/valsartan, beta-blockers, mineralocorticoid receptor antagonists, diuretics, and sodium-glucose cotransporter-2 inhibitors. When clinically indicated antiarrhythmic agents, anticoagu-

Table 1

**Clinical and instrumental characteristics of the 1<sup>st</sup> group of patients**

Parameter	Month 1	Month 12	Month 24
SVEB, %	5.16±0.25	3.20±0.18*	1.80±0.10***
VEB, %	6.80±0.11	2.80±0.07**	1.20±0.05***
NSVT, n (%)	18 (22.5)	11 (13.8)	6 (7.5)
AF, n (%)	14 (17.5)	11 (13.8)	7 (8.8)
SDNN, ms	89.1±8.3	98.3±5.3*	104.9±4.8*
LF/HF	1.17±0.08	1.27±0.07	1.38±0.07*
EDVi, mL/ml	100.8±8.9	82.3±5.8*	75.4±4.3*
LVEF, %	35.2±3.4	43.2±2.8*	49.1±2.9*
GLS, %	6.5±0.6	8.3±0.8*	12.9±0.9**
GCS, %	7.2±0.6	8.6±0.7	11.4±0.9*
Mean NYHA FC, points	3.2	2.8	1.8
6MWT, m	240±18	322±25*	386±29**
LV segments with inflammatory changes, n	4.80±0.27	2.10±0.13**	1.20±0.08***
LV segments with LGE, n	2.25±0.19	2.50±0.21	2.52±0.20
Total number of involved LV segments, n	7.05±0.48	4.60±0.37**	3.72±0.31***
Persistence of inflammation (transition to chronic myocarditis), %	–	49 (61.3)	18 (22.5)
Recovery, n (%)	–	18 (22.5)	42 (52.5)
Transition to DCM, n (%)	–	9 (11.3)	13 (16.3)
Death, n (%)	–	4 (5.0)	7 (8.8)

Differences in the parameters are statistically significant compared with those at the AM onset: \* –  $p < 0.05$ , \*\* –  $p < 0.01$ , \*\*\* –  $p < 0.001$ .

lants, glucocorticoids, and pregabalin in cases of marked anxiety were administered [13, 14]. All patients were evaluated by a psychotherapist. Follow-up examinations of patients were carried out after 12 and 24 months.

Statistical analysis was performed using a computerized database created in Excel XP (Microsoft Office, USA) and processed with Statistica for Windows v.6,0 (StatSoft, USA). Descriptive statistics included the mean (M), standard error of the mean (m), Student's t-value (t), and corresponding p-values. Comparisons between clinical groups were conducted using Student's t-test with statistical significance defined as  $p < 0.05$ . Associations between continuous variables were examined using Pearson's parametric correlation analysis. Cut-off thresholds predicting specific clinical characteristics were determined using Student's criterion within a multivariate regression analysis.

## RESULTS

At the onset of AM patients in the 1<sup>st</sup> group exhibited a substantial burden of both supraventricular and ventricular ectopic activity. Episodes of NSVT were identified in 22,5 % patients, whereas paroxysmal AF was documented in 17,5 % patients, both occurring in the context of impaired HRV as reflected by reduced SDNN and LF/HF values (Table 1). LV dilation (elevated EDVi) together with a decline in LV systolic function (EF, GLS, GCS) took place against the background of a significant number of LV segments with inflammatory changes and the presence of LGE, which determined a high NYHA FC and low tolerance to physical activity based on the 6MWT results.

After 12 months of follow-up the 1<sup>st</sup> group patients demonstrated a marked reduction in the frequency of both ventricular and supraventricular arrhythmias in association with improvements in

Table 2  
**Clinical and instrumental characteristics of the 2<sup>nd</sup> group of patients**

Parameter	Month 1	Month 12	Month 24
Mean NYHA FC, points	2.6	1.9	1.1
6MWT, m	348.0±19.8	388.0±25.0	445.0±29.0**
EDVi, mL/ml	80.9±7.5	75.8±5.4	71.3±4.1*
LVEF, %	44.5±3.8	48.4±3.0	52.5±3.1*
GLS, %	9.26±0.60	11.95±0.78*	13.69±0.76**
GCS, %	9.08±0.67	10.78±0.91	12.71±0.96**
SVEB, %	7.84±0.28	3.80±0.21**	2.10±0.12***
VEB, %	8.40±0.12	2.30±0.08**	1.90±0.06***
NSVT, n (%)	7 (10.9)	5 (7.8)	2 (3.1)
AF, n (%)	9 (14.1)	6 (9.4)	4 (6.3)
SDNN, ms	98.9±7.2	110.2±5.4*	118.7±5.2**
LF/HF	1.21±0.09	1.28±0.07	1.48±0.07*
LV segments with inflammatory lesions, n	3.2±0.12	1.45±0.12**	0.65±0.05***
LV segments with LGE, n	1.21±0.10	1.32±0.11	1.40±0.11
Total number of involved LV segments, n	4.41±0.29	2.77±0.22**	2.05±0.31***
Persistence of inflammation (transition to chronic myocarditis), n (%)	–	23 (36.5 %)	12 (19.1 %)
Recovery, n (%)	–	35 (55.6 %)	40 (63.5 %)
Transition to DCM, n (%)	–	3 (4.8 %)	7 (11.1 %)
Death, n (%)	–	2 (3.2 %)	4 (6.4 %)

Differences in the parameters are statistically significant compared with those recorded at the onset of AM: \* –  $p < 0.05$ , \*\* –  $p < 0.01$ , \*\*\* –  $p < 0.001$ . The clinical course among patients of the 2<sup>nd</sup> group was less severe than that observed in the 1<sup>st</sup> group; 2 and 4 deaths were recorded by 12 and 24 months respectively. Transition to DCM occurred in 3 and 7 patients and recovery was documented in 35 and 42 patients at 12 and 24-month assessment respectively. Persistence of myocardial inflammation consistent with chronic myocarditis was noted in 23 and 12 patients at 12 months and at the end of the follow-up period respectively.

HRV: SDNN increase by 10.3 % ( $p < 0.05$ ) and a trend toward sympathovagal balance improving, indicated by an 8.5 % LF/HF increase. The incidence of NSVT and AF paroxysms remained unchanged, each occurring in 13.8 % of patients. LV systolic function improved substantially with significant increase in LVEF, GLS, and GCS (by 22.7 %, 27.7 % and 19.4 % respectively) together with an 18.4 % EDVi decrease ( $p < 0.05$ ). Cardiac MRI demonstrated a twofold decrease in the number of LV segments with inflammatory involvement while the number of LGE-positive segments remained unchanged suggesting a transformation of LV inflammatory changes into fibrotic. These favorable structural changes was supported by a 34.2 % reduction in total amount of LV myocardial damage ( $p < 0.01$ ), likely contributing to improved LV contractility and decreased LV dilation.

Consistent with these findings the 6MWT distance increased by an average of 34.2 % ( $p < 0.05$ ). At the 24-month follow-up a positive trend towards improvement in the structural and functional state of the heart was maintained: there was 39.5 % LVEF increase and 25.2 % EDVi decrease vs baseline ( $p < 0.01$ ). LV contraction geometry showed marked improvement with GLS increasing by 98 % and GCS – by 58.3 % compared with initial values in the background on a fourfold reduction in the number of LV segments with inflammatory involvement and nearly a twofold decrease in the total number of affected segments. The 6MWT distance increased on average by 60.8 % compared to the debut of AM, which corresponded on average to NYHA FC II. After 24 months 42 patients had fully recovered whereas 18 exhibited persistent myocardial inflammation consistent with

Table 3

**Clinical and instrumental characteristics of the 3<sup>rd</sup> group of patients**

Parameter	Month 1	Month 12	Month 24
Mean NYHA FC, points	1.6	1.2	0.6
6MWT, m	422±12	480±14*	540±16*
EDVi, mL/ml	74.3±3.6	72.1±3.5	68.9±3.3*
LVEF, %	52.3±3.1	55.1±3.2	56.8±3.8
GLS, %	12.7±0.6	14.5±0.8	15.2±0.9*
GCS, %	12.0±0.5	12.9±0.7	13.6±0.7
SVEB, %	12.6±0.4	6.1±0.3**	2.8±0.1***
VEB, %	7.2±0.1	2.5±0.1**	1.2±0.1***
NSVT, n (%)	3 (5.0)	1 (1.67)	–
AF, n (%)	4 (6.67)	3 (5.00)	2 (3.33)
SDNN, ms	112.4±6.4	118.9±5.8	124.5±6.1*
LF/HF	1.47±0.06	1.56±0.08	1.59±0.08*
LV segments with inflammatory involvement, n	2.01±0.06	0.86±0.04**	0.32±0.02***
LV segments with LGE, n	0.81±0.06	0.91±0.07**	0.69±0.06**
Total number of involved LV segments, n	2.81±0.20	1.77±0.13**	1.01±0.08***
Persistence of inflammation (transition to chronic myocarditis), n (%)	–	19 (31.7 %)	8 (13.3 %)
Recovery, n (%)	–	41 (68.3 %)	52 (86.7 %)
Transition to DCM, n (%)	–	–	–
Death, n (%)	–	–	–

Differences in the parameters are statistically significant compared with those recorded at baseline: \* –  $p < 0.05$ , \*\* –  $p < 0.01$ , \*\*\* –  $p < 0.001$ .

chronic myocarditis and showed no meaningful improvement in cardiac structure, function or heart rhythm disturbances. AM was transformed into DCM in 13 patients with deterioration of structural and functional indices despite the absence of active inflammation, accompanied by a marked increase in the volume of fibrotic lesions. During this follow-up period 7 patients died.

The 2<sup>nd</sup> group patients exhibited baseline mild LV dilation and a moderate reduction in LVEF, GLS and GCS (Table 2). Analysis of 24-hour ECG monitoring demonstrated a considerable number of SVEB and VEB, however, in contrast to the 1st group, isolated ectopic complexes predominated occurring in the context of reduced HRV and impaired sympathovagal balance. Cardiac MRI revealed a pronounced degree of inflammatory myocardial involvement, while the extent of fibrotic tissue was lower than that in the 1st group. After 12 months a trend toward improvement in

LVEF was noted, whereas GLS showed a significant increase and GCS exhibited a tendency to rise, with mean changes of 29.1 % and 18.7 %, respectively. Cardiac MRI revealed a more than twofold reduction in the number of LV segments with inflammatory involvement, while the prevalence of LGE-positive (fibrotic) segments remained unchanged. Overall LV involvement decreased 1.6-fold ( $p < 0.01$ ). By 24 months LVEF had restored (> 50 %) with an 11.9 % reduction in EDVi ( $p < 0.05$ ) and further improvement in LV contraction geometry (GLS, GCS). Cardiac MRI continued to demonstrate regression of inflammatory involvement, with a more than twofold reduction in the total number of affected LV segments compared with the initial evaluation. Significant positive dynamics have been registered in terms of reducing the number of VEB and SVEB and further increasing the values of the frequency and spectral parameters of the HRV. Functional capacity improved substantially with the

Table 4

**Comparison of HADS-A and HAM-A anxiety scores and 24-hour ECG results between combatants and civilian patients at the onset of AM**

Parameter	Combatants (n=90)	Civilians (n=114)
<b>HADS-A</b>		
Absence of anxiety, % (n)	31.1 (28)	55.2 (43)
Subclinical anxiety, % (n)	36.7 (33)	25.4 (29)
Clinically manifest anxiety, % (n)	31.1 (28)	19.3 (22)
Mean anxiety score, points	12.65±1.28**	7.67±1.04
<b>HAM-A</b>		
Absent/mild anxiety, % (n)	24.4 (22)	50.8 (58)
Moderate anxiety, % (n)	42.3 (38)	36.9 (42)
Severe anxiety, % (n)	33.3 (30)	12.3 (14)
Mean anxiety score, points	22.12±2.09**	16.67±1.37
<b>24-hour ECG monitoring</b>		
Frequent SVEB, % (n)	18.9 (17)	12.3 (14)
Frequent VEB, % (n)	17.8 (16)	17.5 (20)
NSVT, % (n)	16.6 (15)	15.8 (18)
AF, % (n)	20.0 (18)	11.4 (13)
SDNN, ms	103.2±6.3*	121.0±7.4
LF/HF	1.21±0.09*	1.57±0.10

Differences in the parameters are statistically significant compared with civilian patients: \* –  $p<0.05$ ; \*\* –  $p<0.01$ .

6MWT distance increasing by an average of 27.9 %, corresponding to NYHA FC I.

Patients in the 3<sup>rd</sup> group exhibited the most benign initial course of AM with preserved LVEF without LV dilation, moderate reductions in GLS and GCS and a limited number of LV segments with inflammatory or fibrotic lesions (Table 3). These patients were characterized by predominantly supraventricular rhythm disturbances, with VEB occurring far less frequently. At 12 and 24 months the number of VEB decreased 2,8- and 6-fold respectively, whereas SVEBs decreased 2- and 4,5-fold over the same periods. By the end of the follow-up period the frequency of AF paroxysms had declined by half and no episodes of NSVT were recorded. Throughout both the 12- and 24-month follow-up no deaths or transition to DCM were observed. Persistence of myocardial inflammation consistent with chronic myocarditis was documented in 19 and 8 patients whereas recovery was noted in 41 and 52 patients at 12 and 24 months respectively.

The subsequent stage of the study focused on characterizing key features and conducting a comparative analysis of the clinical presentation and structural-functional parameters of AM in combatants (n=90)

relative to civilian patients (n=114). The groups were comparable in LVEF, EDVi, the extent of inflammatory LV involvement, the mean number of LGE-positive LV segments and NYHA FC. According to the HADS-A and HAM-A results combatants exhibited a higher frequency of both subclinical and clinically manifest anxiety at disease onset compared with civilians, severe anxiety was observed 2.7 times more often among combatants (Table 4). The mean HADS-A anxiety score in combatants was 40.8 % higher than in civilians, while the mean HAM-A score exceeded that of civilians by 24.6 %.

Evaluation of HRV indices and arrhythmic burden at AM onset revealed substantial intergroup differences. The mean daily percentage of SVEB was significantly higher in combatants ( $4.05\pm0.18$  % vs.  $1.77\pm0.23$  % in civilians,  $p<0.01$ ). In addition, AF paroxysms occurred 1,75 times more frequently and frequent SVEB-1,54 times more often in combatants compared with civilians. Combatants demonstrated more pronounced impairment of autonomic heart rhythm regulation, evidenced by a 14.7 % lower SDNN value and a significantly reduced LF/HF ratio which indicated a more pronounced impairment of autonomic regulation

of heart rhythm. No significant between-group differences were observed in the prevalence of ventricular rhythm disturbances, including frequent VEB or episodes of NSVT. At the 12-month ECG follow-up both groups demonstrated a reduction in ventricular and supraventricular rhythm disturbances, along with HRV increase. Combatants continued to show more pronounced sympathovagal imbalance: the LF/HF ratio remained 24.3 % lower suggesting persistent predominance of sympathetic activity. Moreover the mean daily frequency of SVEB remained higher in combatants ( $0.87 \pm 0.08$  % vs.  $0.54 \pm 0.07$  % in civilians,  $p < 0.01$ ). AF paroxysms and frequent SVEB were observed 1,76-fold and 1,69-fold more often in combatants compared with civilians respectively.

According to the results of the correlation analysis performed at the 12-month follow-up combatants demonstrated significant positive associations between the clinically manifest anxiety and the persistence of frequent SVEB ( $r = 0.51$ ,  $p < 0.05$ ) as well as AF paroxysms ( $r = 0.65$ ,  $p < 0.02$ ). Thus combatants who continued to exhibit clinical symptoms of anxiety at 12 months after AM onset were more likely to experience supraventricular rhythm disturbances, including frequent SVEB and AF paroxysms, as a pattern associated with a sympathovagal balance shift toward sympathetic predominance in the context of anxiety disorders. In contrast the persistence of ventricular arrhythmias showed no clear association with the presence of anxiety.

At the final stage of the study, based on the results of the 24-month longitudinal follow-up, multivariate regression models were developed to identify predictors of recovery, persistence of chronic myocarditis and transition to DCM. Recovery was defined in 82 patients (40.4 %) by the absence of clinical symptoms, the absence of rhythm and conduction disturbances on 24-hour ECG monitoring, preserved LVEF ( $\geq 50$  %) and a normal EDVi ( $\leq 75$  mL/m<sup>2</sup>), no evidence of inflammatory LV involvement on cardiac MRI and normal performance on the 6MWT. Persistence of chronic myocarditis in 58 patients (28.5 %) was defined by the presence of inflammatory myocardial changes in  $\geq 3$  LV segments on cardiac MRI at 24 months, reduced/moderately reduced LVEF ( $< 50$  %) and clinical HF manifestation. Transition to DCM in 18 patients (8.8 %) was defined by the absence of inflammatory myocardial changes on cardiac MRI, the presence of LGE in  $\geq 5$  LV segments; reduced LVEF ( $\leq 40$  %) and marked LV dilation ( $EDVi \geq 105$  mL/m<sup>2</sup>) despite optimal medical therapy. Eleven patients (4.4 %) died.

The regression models were appeared as follows:

$$y = a_0 + a_1x_1 + a_2x_2 + \dots + a_nx_n,$$

where  $y$  represents the initial model (recovery, persistence of myocarditis or transition to DCM at 24

months);  $x_1, \dots, x_n$  – denote the independent variables (threshold values of parameters identified during the initial assessment); and  $a_0, \dots, a_n$  – are the model coefficients.

Table 5 presents the regression model parameters. According to the  $\beta$ -coefficients the following baseline parameters demonstrated a significant contribution to the persistence of myocardial inflammation at 24 months:  $EDVi \geq 105$  mL/m<sup>2</sup> ( $\beta = 0.722$ ,  $p_a = 0.023$ );  $LVEF \leq 40$  % ( $\beta = 0.798$ ,  $p_a = 0.034$ );  $GLS \leq 9.0$  % ( $\beta = 0.877$ ,  $p_a = 0.039$ );  $GCS \leq 8.0$  % ( $\beta = 0.419$ ,  $p_a = 0.043$ ); presence of NSVT episodes ( $\beta = 0.522$ ,  $p_a = 0.023$ ); total number of affected LV segments  $\geq 5$  ( $\beta = 0.912$ ,  $p_a = 0.013$ ) and the number of LV segments with inflammatory involvement  $\geq 3$  ( $\beta = 0.460$ ,  $p_a = 0.046$ ). Predictors of transition to DCM included baseline parameters:  $EDVi \geq 105$  mL/m<sup>2</sup> ( $\beta = 0.697$ ,  $p_a = 0.019$ );  $LVEF < 30$  % ( $\beta = 0.608$ ,  $p_a = 0.037$ );  $GLS \leq 8.0$  % ( $\beta = 0.702$ ,  $p_a = 0.030$ );  $GCS \leq 7.0$  % ( $\beta = 0.531$ ,  $p_a = 0.028$ ); total number of affected LV segments  $\geq 9$  ( $\beta = 0.921$ ,  $p_a = 0.009$ ) and the number of LGE-positive LV segments  $\geq 5$  ( $\beta = 0.809$ ,  $p_a = 0.017$ ). Predictors of recovery at 24 months were:  $EDVi \leq 85$  mL/m<sup>2</sup> ( $\beta = 0.645$ ,  $p_a = 0.041$ );  $LVEF \geq 50$  % ( $\beta = 0.730$ ,  $p_a = 0.026$ );  $GLS \geq 11.0$  % ( $\beta = 0.745$ ,  $p_a = 0.008$ );  $GCS \geq 12.0$  % ( $\beta = 0.498$ ,  $p_a = 0.047$ ); total number of affected LV segments  $\leq 3$  ( $\beta = 0.699$ ,  $p_a = 0.038$ ) and the absence of NSVT episodes ( $\beta = 0.521$ ,  $p_a = 0.035$ ) at baseline.

Given the uncertainty in the contemporary international literature and the considerable scientific and clinical interest surrounding patients with AM and intermediate risk our study employed multivariate regression analysis to identify predictors of myocarditis persistence in the 2<sup>nd</sup> group. The corresponding regression model, with parameters presented in Table 6, was appeared as follows:

$$y = a_0 + a_1x_1 + a_2x_2 + \dots + a_nx_n,$$

where  $y$  represents the model initial (persistence of myocarditis at the 24-month follow-up);  $x_1, \dots, x_n$  – denote the independent variables (threshold parameter values identified during the initial evaluation) and  $a_0, \dots, a_n$  – are the model coefficients.

Based on the multivariate analysis results the following baseline parameters were identified as predictors of myocarditis persistence at 24 months in intermediate-risk patients:  $EDVi \geq 95$  mL/m<sup>2</sup> ( $\beta = 0.671$ ,  $p_a = 0.019$ );  $LVEF < 50$  % ( $\beta = 0.863$ ,  $p_a = 0.030$ );  $GLS \leq 11.0$  % ( $\beta = 0.761$ ,  $p_a = 0.022$ ); presence of NSVT episodes ( $\beta = 0.481$ ,  $p_a = 0.034$ ); number of LGE-positive LV segments  $\geq 2$  ( $\beta = 0.922$ ,  $p_a = 0.029$ ) and number of LV segments with inflammatory involvement  $\geq 3$  ( $\beta = 0.851$ ,  $p_a = 0.020$ ).

In addition, multivariate regression models were developed specifically for combatants with AM to enable early prediction of long-term persistence of frequent SVEB ( $\geq 1.0$  % of total daily heartbeats) and AF

Table 5

**Parameters of the multivariate regression models for predicting the clinical course of myocarditis**

Factors (Baseline Parameters)	$\beta$	$S_{\beta}$	$\alpha$	$P_{\alpha}$
<b>Predictors of chronic myocarditis persistence</b>				
EDVi $\geq 105$ mL/m <sup>2</sup>	0.722	0.178	5.721	0.023
LVEF $\leq 40$ %	0.798	0.161	3.412	0.034
GLS $\leq 9.0$ %	0.877	0.160	2.190	0.039
GCS $\leq 8.0$ %	0.419	0.158	3.132	0.043
Presence of NSVT paroxysms	0.522	0.168	2.916	0.023
Total number of affected LV segments $\geq 5$	0.912	0.184	5.007	0.013
LV segments with inflammatory involvement $\geq 3$	0.412	0.139	0.572	0.032
<b>Predictors of AM transition to DCM</b>				
EDVi $\geq 105$ mL/m <sup>2</sup>	0.697	0.152	2.711	0.019
LVEF $< 30$ %	0.608	0.177	2.570	0.037
GLS $\leq 8.0$ %	0.702	0.150	3.299	0.030
GCS $\leq 7.0$ %	0.531	0.150	2.355	0.028
Total number of affected LV segments $\geq 9$	0.921	0.191	6.630	0.009
LGE-positive LV segments $\geq 5$	0.809	0.169	3.988	0.017
<b>Predictors of recovery in patients with AM</b>				
EDVi $\leq 85$ mL/m <sup>2</sup>	0.645	0.149	2.833	0.041
LVEF $\geq 50$ %	0.730	0.174	3.578	0.026
GLS $\geq 11.0$ %	0.745	0.177	3.191	0.008
GCS $\geq 11.0$ %	0.498	0.145	1.987	0.047
Total number of affected LV segments $\leq 3$	0.699	0.156	2.545	0.038
Absence of NSVT episodes	0.521	0.161	2.718	0.035

Table 6

**Parameters of the multivariate regression model predicting myocarditis persistence in patients with intermediate risk**

Factors (baseline parameters)	$\beta$	$S_{\beta}$	$\alpha$	$P_{\alpha}$
<b>Predictors of chronic myocarditis persistence</b>				
EDVi $\geq 95$ mL/m <sup>2</sup>	0.671	0.167	1.984	0.019
LVEF $< 50$ %	0.863	0.181	2.799	0.030
GLS $\leq 11.0$ %	0.761	0.165	1.290	0.022
Presence of NSVT paroxysms	0.481	0.150	0.934	0.034
LGE-positive LV segments $\geq 2$	0.922	0.171	2.432	0.029
LV segments with inflammatory involvement $\geq 3$	0.851	0.170	1.981	0.020

Table 7

**Parameters of the multivariate regression models predicting the clinical course of myocarditis in combatants**

Factors (baseline parameters)	$\beta$	$S_{\beta}$	$\alpha$	$P_{\alpha}$
<b>Predictors of frequent SVEB persistence</b>				
HADS-A score $\geq 11$ points	0.455	0.153	1.109	0.035
HAM-A score $\geq 25$ points	0.514	0.168	1.275	0.022
LF/HF $\leq 1.20$	0.610	0.138	0.801	0.044
Total number of affected LV segments $\geq 5$	0.745	0.171	1.490	0.025
LGE-positive LV segments $\geq 2$	0.809	0.173	1.709	0.027
<b>Predictors of persistent AF paroxysms</b>				
EDVi $\geq 105$ mL/m <sup>2</sup>	0.755	0.164	1.819	0.023
LVEF $\leq 40$ %	0.951	0.168	2.654	0.028
HADS-A score $\geq 11$ points	0.411	0.138	0.774	0.048
HAM-A score $\geq 25$ points	0.498	0.155	1.009	0.045
LF/HF $\leq 1.20$	0.547	0.161	1.109	0.027
Total number of affected LV segments $\geq 5$	0.981	0.179	2.897	0.006
LGE-positive LV segments $\geq 2$	0.880	0.174	2.185	0.026

paroxysms, the parameters are presented in *Table 7*. In general, the regression models were appeared as follows:

$$y = a_0 + a_1x_1 + a_2x_2 + \dots + a_nx_n,$$

where  $y$  represents the model initial (presence of frequent SVEB or AF paroxysms at the 24-month follow-up);  $x_1, \dots, x_n$  – denote the independent variables (threshold parameters values identified during the initial evaluation); and  $a_0, \dots, a_n$  – the model coefficients.

Thus, in combatants with AM, the following baseline parameters were identified as predictors of the persistence of frequent SVEB at 24 months: HADS-A score  $\geq 11$ ; HAM-A score  $\geq 25$ ; LF/HF ratio  $\leq 1.20$ ; total number of affected LV segments  $\geq 5$  and  $\geq 2$  LGE-positive LV segments. Predictors of the persistence of AF paroxysms at 24 months included: LV EDVi  $\geq 105$  mL/m<sup>2</sup>; LVEF  $\leq 40$  %; HADS-A score  $\geq 11$ ; HAM-A score  $\geq 25$ ; LF/HF ratio  $\leq 1.20$ ; total number of affected LV segments  $\geq 5$  and  $\geq 2$  LGE-positive LV segments.

## DISCUSSION

Despite substantial advances in diagnostic modalities and pharmacotherapy, prognostic assessment in AM remains a highly relevant and challenging clinical issue [15]. Identifying patients at increased risk of adverse cardiovascular events is essential for optimizing follow-up strategies and enabling timely adjust-

ment of therapeutic interventions. A key component of risk stratification is the detection of early prognostic markers that can signal a likelihood of a complicated disease course, facilitate identification of high-risk individuals, and guide the need for intensified surveillance and pharmacotherapy.

We conducted the first study in Ukraine in accordance with the most recent ESC guidelines, incorporating current achievements on the diagnosis and management of patients with myocarditis taking into account stratification by risk profile at initial presentation and evaluate the long-term outcomes with particular attention to those categorized as intermediate risk [1, 16]. A total of 204 patients were assigned to high, intermediate or low risk groups at AM onset. In 2016 D. Sinagra and colleagues proposed a risk-stratification framework for patients with clinically suspected myocarditis, based on three clinical phenotypes – myocarditis with low, intermediate, and high risk. This model was subsequently integrated and further refined in the ESC 2025 recommendations [1, 17]. Each risk category is associated with distinct prognostic implications and requires tailored approaches to patient monitoring.

The results of our study substantially expand current understanding of the spectrum of early prognostic markers associated with the clinical course of myocarditis and may facilitate timely risk stratification in affected patients. To date it has been well established

that reduced LVEF, LV dilation, the presence of LGE on cardiac MRI and ventricular arrhythmias are all associated with an poor prognosis in patients with myocarditis [5, 18, 19]. Our results are consistent with reports from international investigators highlighting the high clinical value of multiparametric risk-assessment tools for predicting adverse outcomes and guiding prognosis in myocarditis [20, 21].

The novelty of our study lies in demonstrating the statistically significant predicting value of the clinical course of myocarditis for LV GLS and GCS, the extent of inflammatory and fibrotic myocardial involvement (LGE), and the total number of affected LV segments. Each of these markers showed a clear association with specific patterns of disease evolution. Our results align with findings reported in other contemporary studies. Speckle-tracking EchoCG plays a key role in the early detection of subclinical myocardial dysfunction and enables quantitative assessment of myocardial deformation, GLS and GCS are highly sensitive markers of early contractile impairment even when LVEF remains preserved [22]. In myocarditis a reduction in GLS reflects inflammatory activity and the extent of myocardial involvement, and its improvement over time correlates with clinical recovery. Incorporating speckle-tracking EchoCG into the comprehensive evaluation of patients with myocarditis enhances the accuracy of risk stratification, facilitates timely detection of relapse or persistent inflammation and supports assessment of therapeutic effectiveness [1]. The extent and temporal evolution of LGE have major prognostic implications in patients with myocarditis. Persistence or progression of LGE beyond the acute phase reflects ongoing inflammation and fibrotic remodeling, which is associated with an increased risk of HF and life-threatening arrhythmias [19, 23]. Evidence from other studies indicates that the combined use of T1/T2 mapping and LGE imaging not only confirms the presence of myocarditis but also allows quantification of the degree of fibro-inflammatory myocardial injury, which directly correlates with clinical prognosis [24, 25]. Modern cardiac MRI is increasingly evolving from a purely diagnostic tool into a powerful instrument for risk stratification, aligning with the principles of personalized cardiology. Follow-up MRI performed at 6–12 months provides essential information on therapeutic response and disease trajectory, underscoring the importance of longitudinal monitoring.

An additional important achievement of our study was the identification of predictors of persistent supraventricular arrhythmias and their association with anxiety symptoms in combatants with AM, a finding of particular relevance under current conditions for guiding timely and individualized treatment strategies.

Anxiety disorders and chronic emotional stress lead to hyperactivation of the sympathetic-adrenal system, reduced HRV, and elevated levels of pro-inflammatory cytokines, creating a milieu that promotes arrhythmogenesis and contributes to the progression of cardiac pathology [26]. Disturbances in autonomic regulation of cardiac function represent one of the key pathophysiological mechanisms underlying the progression of myocarditis and the development of its complications. Exposure to combat-related stress is associated with increased anxiety, sleep disturbances, and signs of post-traumatic maladaptation, which are leading to persistent autonomic cardiac [27]. Chronic sympathetic activation, together with depletion of parasympathetic regulatory capacity, may serve as a pathogenic driver of arrhythmogenic substrate formation, particularly in the presence of inflammatory or fibrotic myocardial involvement.

According to the findings of S. Perek et al. [28], impaired HRV, specifically reduced SDNN, in patients with myocarditis was a significant predictor of adverse clinical outcomes and an increased risk of arrhythmias during 6-month follow-up. When combined with cardiac MRI data, where the presence of LGE reflects the extent of fibrosis and tissue remodelling, HRV serves as a functional risk marker that complements morphological assessment. Previous studies have demonstrated that patients with LGE exhibit markedly higher rates of ventricular arrhythmias and the combination of LGE with a low SDNN value ( $< 100$  ms) increases the predicted risk of sudden cardiac death more than threefold [7, 19]. Thus an integrated assessment that includes evaluation of psychological status, HRV and imaging-derived structural and functional cardiac characteristics in combatants with AM enables the identification of individuals at heightened arrhythmogenic risk. This approach is essential for personalized risk stratification and for optimizing therapeutic management in this vulnerable patient population.

Our results reinforce the value of risk-based stratification in patients with AM as a practical approach for anticipating the clinical course of the disease. The prognostic markers identified in this study enable early detection of individuals at increased risk for adverse outcomes, support timely therapeutic adjustment, and enhance the effectiveness of long-term management. Furthermore our work represents the first Ukrainian study to perform a comparative assessment of structural-functional cardiac parameters, HRV indices and heart rhythm disturbances in combatants in comparison with civilian patients. The analysis underscored the contribution of stress-related factors as triggers for the onset and persistence of arrhythmia with their impact modulated by the psychological profile of the patient.

## CONCLUSIONS

1. Predictors of AM persistence over a 24-month follow-up period were identified according to multi-variable regression analysis. Transition to chronic myocarditis was predicted by the following baseline parameters: LV EDVi  $\geq 105$  mL/m<sup>2</sup>, LVEF  $\leq 40$  %, GLS  $\leq 9.0$  %, GCS  $\leq 8.0$  %, NSVT paroxysms, total number of affected LV segments  $\geq 5$  and  $\geq 3$  LV segments with inflammatory lesions. Transition to DCM was associated with an EDVi  $\geq 105$  mL/m<sup>2</sup>, an LVEF  $< 30$  %, GLS  $\leq 8.0$  %, GCS  $\leq 7.0$  %, a total number of affected segments  $\geq 9$  and  $\geq 5$  segments with LGE. Predictors of recovery included EDVi  $\leq 85$  mL/m<sup>2</sup>, LVEF  $\geq 50$  %, GLS  $\geq 11.0$  %, GCS  $\geq 12.0$  %, a total of  $\leq 3$  affected LV segments and the absence of NSVT paroxysms during the initial assessment.

2. Current approaches to prognostic stratification of the clinical course of myocarditis in patients at inter-

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## References

- Schulz-Menger J, Collini V, Gröschel J, et al. 2025 ESC Guidelines for the management of myocarditis and pericarditis. *Eur Heart J*. 2025;00:1-90. <https://doi.org/10.1093/eurheartj/ehaf192>
- Grün S, Schumm J, Greulich S, Wagner A, Schneider S, Bruder O, Kispert EM, Hill S, Ong P, Klingel K, Kandolf R, Sechtem U, Mahrholdt H. Long-term follow-up of biopsy-proven viral myocarditis: predictors of mortality and incomplete recovery. *J Am Coll Cardiol*. 2012 May 1;59(18):1604-15. <https://doi.org/10.1016/j.jacc.2012.01.007>
- Kindermann I, Kindermann M, Kandolf R, et al. Predictors of outcome in patients with suspected myocarditis. *Circulation*. 2008;118:639-48. <https://doi.org/10.1161/CIRCULATIONAHA.108.769489>
- Grossman SM, Pravda NS, Orvin K, Hamdan A, Vaturi M, Bengal T, Kornowski R, Weissler-Snir A. Characterization and long-term outcomes of patients with myocarditis: a retrospective observational study. *Postepy Kardiologii Interwencyjnej*. 2021 Mar;17(1):60-7. <https://doi.org/10.5114/aic.2021.104770>
- Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, et al. Comparison of original and 2018 Lake Louise criteria for diagnosis of acute myocarditis. *Radiol Cardiothoracic Imaging*. 2020;2(3): e190010. <https://doi.org/10.1148/ryct.2019190010>
- Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, et al. Cardiovascular magnetic resonance in nonischemic myocardial inflammation: Expert recommendations. *J Am Coll Cardiol*. 2018; 72(24):3158-76. <https://doi.org/10.1016/j.jacc.2018.09.072>
- Gräni C, Eichhorn C, Büre L, Murthy VL, Agarwal V, Kaneko K, et al. Prognostic value of cardiac magnetic resonance tissue characterization in risk stratifying patients with suspected myocarditis. *JACC*. 2017; 70(16):1964-76. <https://doi.org/10.1016/j.jacc.2017.08.050>
- Kovalenko VM, Nesukay EG, Cherniuk SV, Kozliuk AS, Kyrychenko RM. Diahnostyka ta likuvannia miokardytu. Rekomendatsii Vseukrainskoi asotsiatsii kardiologiv Ukrainy. *Ukrainskyi kardiologichnyi zhurnal*. 2021;28(3):67-88. Ukraine. <https://doi.org/10.31928/1608-635X-2021.3.6788>
- Cheng Z, Li-Sha G, Yue-Chun L. Autonomic nervous system in viral myocarditis: Pathophysiology and therapy. *Curr Pharm Des*. 2016;22:485-98. <https://doi.org/10.2174/1381612822666151222160810>
- Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2015;28:1-39.e14. <https://doi.org/10.1016/j.echo.2014.10.003>
- Amzulescu MS, De Craene M, Langet H, et al. Myocardial strain imaging: review of general principles, validation, and sources of discrepancies. *Eur Heart J Cardiovasc Imaging*. 2019;20:605-19. <https://doi.org/10.1093/ehjci/jez041>
- Christensen AV, Dixon JK, Juel K, Ekholm O, Rasmussen TB, Borregaard B, et al. Psychometric properties of the Danish Hospital Anxiety and Depression Scale in patients with cardiac disease: results from the DenHeart survey. *Health Qual Life Outcomes*. 2020;18:9. <https://doi.org/10.1186/s12955-019-1264-0>
- McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by

- the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) With the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J*. 2021;42(36):3599-726. <https://doi.org/10.1093/eurheartj/ehab368>
14. Nesukay EG, Cherniuk SV, Talaieva TV, Kirichenko RM, Slyvna AB, Titov IYu, Adaricheva ZhG, Nesukai VG. Diagnostika ta medikamentozna korektsiya trivogi u patsiyentiv iz sertsevo-sudinnimi zahvoryuvannyami: dosvid odnogo tsentru. *Ukr Med Chasopys*. 2025;4(170):IV-V:73-78 <https://doi.org/10.32471/umj.1680-3051.266837>
  15. Tschöpe C, Ammirati E, Bozkurt B, Caforio AL, Cooper LT, Felix SB, et al. Myocarditis and inflammatory cardiomyopathy: current evidence and future directions. *Nat Rev Cardiol*. 2021;18(3):169-93. <https://doi.org/10.1038/s41569-020-00435-x>
  16. Ammirati E, Frigerio M, Adler ED, Basso C, Birnie DH, Brambatti M, et al. Management of Acute Myocarditis and Chronic Inflammatory Cardiomyopathy: An Expert Consensus Document. *Circ Heart Fail*. 2020 Nov;13(11):e007405. <https://doi.org/10.1161/CIRCHEARTFAILURE.120.007405>
  17. Sinagra G, Anzini M, Pereira NL, Bussani R, Finocchiaro G, Bartunek J, et al. Myocarditis in clinical practice. *Mayo Clin Proc*. 2016;91(9):1256-66. <https://doi.org/10.1016/j.mayocp.2016.05.013>
  18. Peretto G, Sala S, Rizzo S, De Luca G, Campochiaro C, Sartorelli S, et al. Arrhythmias in myocarditis: state of the art. *Heart rhythm*. 2019;16(5):793-801. <https://doi.org/10.1016/j.hrthm.2018.11.024>
  19. Mahrholdt H, Greulich S. Prognosis in myocarditis: better late than never! *J Am Coll Cardiol*. 2017;70(16):1988-90. <https://doi.org/10.1016/j.jacc.2017.08.062>
  20. Ediger DS, Brady WJ, Koyfman A, Long B. High risk and low prevalence diseases: Myocarditis. *Am J Emerg Med*. 2024 Apr;78:81-8. <https://doi.org/10.1016/j.ajem.2024.01.007>
  21. Di Lisi D, Madaudo C, Macaione F, Scelfo D, Alaimo C, Gargano M, Marotta A, Giardina O, Novo G. Myocarditis prognostic score: a new risk assessment tool in patients with myocarditis. *Eur Heart J*. 2024 Oct;45(Suppl 1):ehae666.159. <https://doi.org/10.1093/eurheartj/ehae666.159>
  22. Thomas JD, Edvardsen T, Abraham T, Appadurai V, Badano L, et al. Clinical Applications of Strain Echocardiography: A Clinical Consensus Statement From the American Society of Echocardiography Developed in Collaboration With the European Association of Cardiovascular Imaging of the European Society of Cardiology. *J Am Soc Echocardiogr*. 2025 Aug 26:S0894-7317(25)00395-5. <https://doi.org/10.1016/j.echo.2025.07.007>
  23. Kuruvilla S, Adenaw N, Katwal AB, Lipinski MJ, Kramer CM, Salerno M. Late gadolinium enhancement on cardiac magnetic resonance predicts adverse cardiovascular outcomes in nonischemic cardiomyopathy: a systematic review and meta-analysis. *Circulation: Cardiovascular Imaging*. 2014;7(2):250-8. <https://doi.org/10.1161/CIRCIMAGING.113.001144>
  24. Habib G, Lurz P, Puntmann VO. Cardiac magnetic resonance in inflammatory heart disease: current applications and future perspectives. *Heart Fail Rev*. 2024;29(5):1321-35. <https://doi.org/10.1007/s44326-024-00026-x>
  25. Verma M, Kumar A, Thakur AK, Singh R, Kadiwar M, Prasad R, et al. Diagnostic performance of cardiovascular magnetic resonance parametric mapping as per modified Lake Louise Criteria in acute myocarditis. *Indian Heart J*. 2025;77(1):14-21. <https://doi.org/10.1016/j.ihj.2025.01.004>
  26. Celano CM, Daunis DJ, Lokko HN, Campbell KA, Huffman JC. Anxiety disorders and cardiovascular disease. *Curr Psychiatr Rep*. 2016;18(11):101. <https://doi.org/10.1007/s11920-016-0739-5>
  27. Pavlova I, Graf-Vlachy L, Petrytsa P, Wang S, Zhang SX. Early evidence on the mental health of Ukrainian civilian and professional combatants during the Russian invasion. *Eur Psychiatr*. 2022;65(1):79. <https://doi.org/10.1192/j.eurpsy.2022.2335>
  28. Perek S, Nussinovitch T, Cohen R, Gidron Y, Raz Pasteur A. Ultra-shortheart rate variability predicts clinical outcomes in patients with a clinical presentation consistent with myocarditis. *J Clin Med*. 2023;12:89. <https://doi.org/10.3390/jcm12010089>

## Характеристика та прогнозування перебігу гострого міокардиту в пацієнтів в умовах воєнного стану в Україні: результати довгострокового спостереження

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**Мета роботи** – дослідити перебіг міокардиту в пацієнтів, зокрема військовослужбовців, з різними профілями ризику та визначити прогностичні чинники несприятливих наслідків протягом 24 місяців спостереження в умовах воєнного стану.

**Матеріали і методи.** Обстежено 204 пацієнти (134 чоловіки та 70 жінок) з гострим міокардитом (ГМ), серед них було 90 військовослужбовців. Вік пацієнтів становив у середньому (38,2±8,6) року. Пацієнти були розподілені на 3 групи: 1-шу становили 80 осіб з високим ризиком, які мали виявлену *de novo* знижену фракцію викиду (ФВ) лівого шлуночка (ЛШ) – ≤ 40 %, прояви серцевої недостатності та розповсюджене відстрочене контрастування при магнітно-резонансній томографії (МРТ) серця; 2-гу – 64 хворих з помірним ризиком з помірно зниженою ФВ ЛШ (41–49 %) та ураженням ≥ 2 сегментів ЛШ при МРТ серця; 3-тю – 60 пацієнтів з низьким ризиком із ФВ ЛШ ≥ 50 % та ураженням < 2 сегментів ЛШ при МРТ серця. Всім пацієнтам проводили ехокардіографію зі спекл-трекінг методи-

кою, добове моніторування ЕКГ з аналізом порушень серцевого ритму та варіабельності ритму серця, МРТ серця з відстроченим контрастуванням. Обстеження виконували в дебюті ГМ, через 12 та 24 міс спостереження.

**Результати та обговорення.** Встановлено предиктори персистенції міокардиту протягом 24 міс: індекс кінцеводіастолічного об'єму ЛШ  $\geq 105$  мл/м<sup>2</sup>; величина ФВ ЛШ  $\leq 40$  %; показник поздовжньої глобальної деформації ЛШ  $\leq 9,0$  %; показник циркулярної глобальної деформації ЛШ  $\leq 8,0$  %; наявність пароксизмів нестійкої шлуночкової тахікардії; загальна кількість уражених сегментів ЛШ  $\geq 5$  та кількість сегментів ЛШ із наявністю запальних змін  $\geq 3$ , визначені на початку ГМ. Предикторами трансформації захворювання в дилатаційну кардіоміопатію були: індекс кінцеводіастолічного об'єму ЛШ  $\geq 105$  мл/м<sup>2</sup>; ФВ ЛШ  $< 30$  %; показник поздовжньої глобальної деформації ЛШ  $\leq 8,0$  %; показник циркулярної глобальної деформації ЛШ  $\leq 7,0$  %; загальна кількість уражених сегментів ЛШ  $\geq 9$ , а також кількість сегментів ЛШ із наявністю відстроченого контрастування  $\geq 5$ . Предикторами одужання через 24 міс були: індекс кінцеводіастолічного об'єму ЛШ  $\leq 85$  мл/м<sup>2</sup>; ФВ ЛШ  $\geq 50$  %; показник поздовжньої глобальної деформації ЛШ  $\geq 11,0$  %; показник циркулярної глобальної деформації ЛШ  $\geq 12,0$  %; загальна кількість уражених сегментів ЛШ  $\leq 3$ , а також відсутність пароксизмів шлуночкової тахікардії при ініціальному обстеженні. У військовослужбовців з міокардитом встановлено предиктори персистенції частоті надшлуночкової екстрасистолії та пароксизмів фібриляції передсердь через 24 міс спостереження: наявність  $\geq 11$  балів тривоги за шкалою HADS-A та  $\geq 25$  балів за шкалою HAM-A, співвідношення LF/HF  $\leq 1,20$ , загальна кількість уражених сегментів ЛШ  $\geq 5$ , кількість сегментів ЛШ із наявністю відстроченого контрастування  $\geq 2$ , визначені при ініціальному обстеженні; додатковими предикторами персистенції пароксизмів фібриляції передсердь також були: індекс кінцеводіастолічного об'єму ЛШ  $\geq 105$  мл/м<sup>2</sup>, фракція викиду ЛШ  $\leq 40$  %.

**Висновки.** За результатами проведених досліджень удосконалено сучасні підходи до прогнозування перебігу міокардиту різного ризику і встановлено предиктори одужання, довготривалої персистенції захворювання та його трансформації в дилатаційну кардіоміопатію. У військовослужбовців з міокардитом встановлено предиктори персистенції частоті надшлуночкової екстрасистолії і пароксизмів фібриляції передсердь через 24 міс спостереження та виявлено зв'язок цих аритмій з наявністю тривоги і порушенням варіабельності ритму серця.

**Ключові слова:** міокардит, дисфункція лівого шлуночка, аритмії, візуалізація, варіабельність ритму серця, прогноз, дилатаційна кардіоміопатія, військовослужбовці