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Original Research Article

Clinical, laboratory and echocardiographic features of patients with ST-elevation myocardial infarction complicated by ventricular arrhythmias: an observational study

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ABSTRACT

Background: Early malignant ventricular arrhythmias (VAs) during ST-elevation myocardial infarction (STEMI) significantly contribute to in-hospital mortality but appear to have little impact on long-term prognosis, likely because the arrhythmic substrate is reversed after revascularization.
Methods: The study included 91 patients with STEMI who were admitted to the Grodno State Cardiological Center for treatment between February 2024 and February 2025. Group 1 included 64 patients with STEMI, whereas Group 2 included 29 patients with STEMI and ventricular arrhythmias (sustained VT or VF). All patients underwent clinical, laboratory, and instrumental examinations, including coronary angiography. Statistical analyses were performed using STATISTICA 12.0 software.
Results: Among 29 patients with VAs, six had ventricular fibrillation treated with electrical cardioversion, three had sustained VT, and 20 had episodes of non-sustained VT. According to the results of coronary angiography, 24 patients had undergone percutaneous coronary intervention, and five patients had stenosis of the left main coronary artery; therefore, they underwent coronary artery bypass graft operation. Patients with STEMI and ventricular tachyarrhythmias were characterized by a higher prevalence of inferior localization of MI (p=0.001), higher WBCs counts (0.008), higher glucose levels (p=0.008), higher troponin levels (p=0.004), lower left ventricular ejection fraction in both M- and B-modes (p=0.001), and higher contractility index (p=0.001).
Conclusions: These results demonstrates that inflammatory markers, myocardial injury, compromised ventricular function, and metabolic disruptions depict that the pathophysiology of arrhythmias in STEMI patients, highlighting the significance of early observation and timely treatment program to decrease the arrhythmic risk.

Keywords: Left ventricular ejection fraction, ST-elevation myocardial infarction, Troponin, Ventricular arrhythmias, Ventricular tachycardia

INTRODUCTION

Acute coronary syndrome (ACS) is a major cause of morbidity and mortality globally. The incidence of this disease has markedly increased over the past few decades. There are various factors that can be attributed to this propagation including an aging population, improved survival rates in coronary artery disease patients, and increased sensitivity of biomarkers (mainly troponin).[1,2]

Ventricular fibrillation (VF) and sustained ventricular tachycardia (VT) are potentially fatal complications that develop in approximately 5% of patients with ST-elevation myocardial infarction (STEMI). Particularly,

these arrhythmias can cause unstable hemodynamics or even deteriorate up to very severe forms of electrical instability which leads to sudden cardiac death.[1-3]

The pathophysiology of ventricular arrhythmias coexisting with STEMI is complex. Following ST elevation acute coronary syndrome (STE-ACS), cardiac muscles undergo remarkable remodeling at the cellular, molecular, genetic, and interstitial levels. This remodeling process involves various mechanisms, such as cell death and protein changes, which involve contraction, necrosis, and fibrosis, leading to anatomical and functional changes in the heart. The development of the fibrotic connective tissue in the cardiac muscles substantially complicating the transmission of the electrical impulses not only due to its arrhythmogenic potential and also for properties which decreases the nature of contractility in these tissues.[4]

Ventricular tachycardia is a frequent arrythmia in acute MI settings, mainly connected with widespread ventricular disease, decreased left ventricular function, and ventricular aneurysm.[5] Clinical manifestations may change, consisting of mild palpitations to signs of decreased cardiac output, particularly dyspnea, lightheadedness and syncope.[6]

According to the guidelines, the treatment of STE-ACS patients with ventricular arrhythmia underscores the significance of percutaneous coronary intervention (PCI) following immediate reperfusion therapy, which is the gold standard performed within a time period of 12 h of symptom onset. In cases where PCI cannot be performed within 90 min, fibrinolysis is the alternative strategy.[7,8]

Prompt revascularization and comprehensive drug therapy (beta-blockers, ACE inhibitors, and anti-platelets) have remarkably reduced the occurrence of ventricular arrhythmias. Nearly 10% of patients who survive myocardial infarction have a high mortality rate during the first months to years. About 50% of all deaths among these high-risk patients are due to sudden death caused by sustained ventricular arrhythmias.[7]

It is very important to understand the clinical, laboratory, and echocardiographic features of patients with STE-ACS with VA. These features can be used to identify high-risk patients who may require more intensive monitoring and management. They may help make therapeutic decisions and risk stratification and understand the mechanisms of arrhythmogenesis in the acute MI setting.

This study aimed to identify the clinical, anamnestic, laboratory, and echocardiographic features of patients with STEMI and ventricular arrhythmias compared to those with uncomplicated STEMI.

METHODS

This analytical observational study with a case-control study design included 91 patients with STEMI who were admitted to the Grodno Regional Clinical Cardiological Center for treatment between February 2024 and February 2025. The inclusion criteria were: patients with acute STEMI, age >18 years and agreement to participate in the study. Group 1 included 64 patients with uncomplicated STEMI, whereas Group 2 included 29 patients with STEMI and ventricular arrhythmias (sustained VT or VF). The exclusion criteria were non-STEMI, chronic rheumatic heart disease, acute myocarditis, valvular pathology of the heart requiring surgical correction, prosthetic heart valves, oncological diseases, and severe concomitant extracardiac pathology. All patients underwent clinical, laboratory, and instrumental examinations, including transthoracic echocardiography and coronary angiography.

Echocardiography was performed using a Phillips iE33 device with a multi-frequency sensor (frequency 2.5-5.0 MHz). The examination was performed with the patient lying on his left side with his back to the researcher or on his back in the supine position. The study protocol included the following indicators: LA and right atrium (RA) diameter in 2-chamber and 4-chamber mode, end-systolic diameter and end-diastolic diameter (mm) of the left ventricle (LV), LVEF, assessment of the state of the valvular apparatus of the heart, and degree of regurgitation on the valves.

All patients underwent coronary angiography according to the Judkins method (1967) in the X-ray operating room using Philips Azurion 7 and GE Innova 3100 IQ angiographic units. The computer program of the GE Innova 3100 IQ unit was used for the quantitative assessment of the stenoses.

Statistical analysis was performed using the STATISTICA 12.0 software package with a preliminary check for normal distribution using a distribution histogram. Quantitative data with a non-normal distribution are presented as medians and 25% and 75% quartiles. As most quantitative characteristics did not obey the normal distribution law, non-parametric methods were used for comparison. The Mann-Whitney U test was used to assess differences in quantitative traits between two independent groups. At a significance level of p less than 0.05, it was believed that the studied indicator in the compared groups had statistically significant differences.

The study was performed in accordance with Good Clinical Practice standards and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before their inclusion in the study.

RESULTS

The clinical characteristics of the patients are shown in Table 1. Patients in both groups were comparable in age (58 [52; 64] vs. 60 [55; 64], p=0.181) and sex (male 78% vs. 76%, p=0.865). Group 1 had a significantly higher

prevalence of obesity (p=0.038), particularly class 1 obesity (p=0.045), and a greater incidence of hypertension (p=0.006) than Group 2. The distribution of MI localization also differed significantly, with Group 1 more frequently experiencing anterior-lateral MI, while Group 2 had a higher occurrence of inferior MI (p<0.05).

Table 1: Clinical characteristics of patients (Me [25%;75%]).

Parameters	Group 1	Group 2	P value
Male gender, N (%)	50 (78)	22 (76)	>0.05
Age, years	57.7 [52;64]	60.4 [55; 64]	>0.05
Body mass index, kg/m	23.2 [26.8; 32.1]	28.4 [25.1; 31.2]	0.05
Obesity, N (%)	30 (46.8)	7 (24.1)	0.038
Class 1, N (%)	20 (31.2)	4 (13.8)	0.045
Class 2, N (%)	8 (12.5)	3 (10.3)	>0.05
Class 3, N (%)	2 [3.125)	-	>0.05
Stage 1, N (%)	13 (20.3)	7 (24.1)	>0.05
Stage 2, N (%)	41 (64.1)	14 (48.3)	>0.05
Stage 3, N (%)	7 (10.9)	1 (3.5)	>0.05
Hypertension, N (%)	61 (95.3)	22 (75.9)	0.006
Localization of MI			
Anterior, N (%)	33 (51.6)	14 (48.3)	>0.05
Inferior, N (%)	11 (17.2)	12 (41.4)	0.001
Lateral, N (%)	11 (17.2)	3 (10.3)	>0.05
Anterior-lateral, N (%)	9 (14.1)	-	0.034
Diabetes mellitus, N (%)	13 (20.3)	3 (10.3)	>0.05
Atrial fibrillation, N (%)	-	4 (13.8)	0.003
Atrial flutter, N (%)	-	1 (3.5)	>0.05

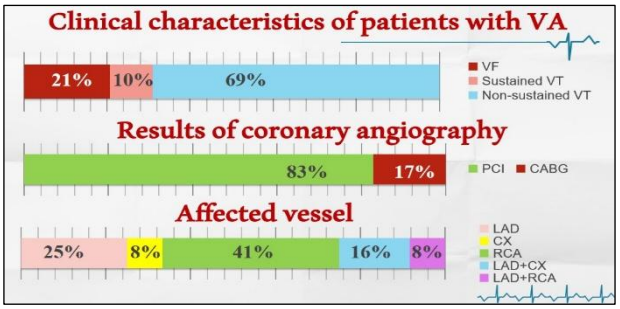

Figure 1: Clinical characteristics of patients with Vas.

Among 29 patients with VAs, 6 - had ventricular fibrillation treated by electrical cardioversion, 3 had sustained VT, and 20 had episodes of non-sustained VT (see Figure 1). According to the results of coronary angiography, 24 patients had undergone PCI and 5 patients had stenosis of the left main coronary artery, therefore they underwent CABG operation. All surgical interventions were performed without complications. Among the 24 patients who underwent PCI, 10 had stenosis of the right coronary artery, 6 had stenosis of the left anterior descending artery, and 2 had stenosis of the circumflex artery. The remaining patients had two of these three arteries affected.

The laboratory parameters of the patients are presented in Table 2. Laboratory parameters showed that patients with VAs had notably higher WBC counts (p=0.008), suggesting an inflammatory response. Also, patients with ventricular arrhythmias had significantly higher levels of urea (6.1 [4.8; 7.8] vs 5.2 [3.9; 6.1] mmol/L, p=0.04), and glucose (7.9 [5.3; 8.8] vs 7.1 [5.3; 8.1] mmol/L, p=0,008). Patients with VAs had higher urea and glucose, pointing to potential metabolic disturbances. In addition, patients in Group 2 had significantly higher troponin levels (30430 [6352; 50000] vs. 12479 [846; 15964] ng/L, p=0.004) than those in Group 1, indicating potentially greater myocardial injury. Other laboratory parameters, including lipid profile parameters (total cholesterol, triglycerides, LDL, and HDL), electrolytes, and liver enzymes, were comparable (p>0.05). The echocardiographic parameters of the patients are presented in Table 3.

Table 2: Laboratory parameters of patients (Me [25%;75%]).

Parameters	Group 1	Group 2	P value
RBC, 10^{12}/L	4.8 [4.48; 5.13]	4.7 [4.4; 5]	>0.05
Age, years	57.7[52;64]	60.4[55; 64]	>0.05
Hemoglobin, g/L	151 [144; 161]	147 [140; 157]	>0.05
WBC, 10^9/L	9.80 [7.6; 11.5]	12.25 [9.3; 15.5]	0.008
ESR, mm/h	8.7 [3.5; 11]	7.1 [4; 9]	>0.05
Platelets, 10^9/L, N (%)	8 (12.5)	3 (10.3)	>0.05
Urea, mmol/L	5.2 [3.9; 6.1]	6.1 [4.8; 7.8]	0.04
Creatinine, µmol/L	75.5[64.9; 80.5]	81.9 [68; 88]	>0.05
eGFR, ml/min/1.73m^2	90.3 [78.7; 104.9]	82.1[62; 100]	>0.05
Cholesterol, mmol/L, N (%)	7 (10.9)	1 (3.5)	>0.05
Glucose, mmol/L	7.2 [5.3;8.1]	8.8 [6.0; 10.0]	0.008

Continued.

Parameters	Group 1	Group 2	P value
LDL, mmol/L	1.05 [0.85; 1.18]	2.30 [1.7; 2.85]	>0.05
HDL, mmol/L	1.05 [0.85; 1.18]	1.01 [0.81; 1.12]	>0.05
Triglycerides, g/L	2.03 [1.26; 1.83]	1.17 [0.87; 1.38]	>0.05
AST	70.1 [22.6; 87]	74.5 [24.9; 109]	>0.05
ALT	39.7 [23; 48.8]	51.8 [22.5; 78.1]	>0.05
Sodium, mEq/L	140.6 [140;142.175]	140.7 [139.6; 143]	>0.05
Potassium, mEq/L	4.37 [4; 4.6]	4.19 [3.9; 4.4]	>0.05
Troponin, ng/L	12479 [846; 15964]	30431 [6352; 50000]	0.004

Abbreviations: RBC, red blood cells; WBC, white blood cells; ESR, erythrocyte sedimentation rate; eGFR, estimated glomerular filtration rate; LDL, low-density lipoprotein; HDL, high-density lipoprotein; AST, aspartate aminotransferase; ALT, alanine aminotransferase

Table 3: Echocardiographic parameters of patients (Me [25%;75%]).

Parameter	Group 1	Group 2	P value
LA diameter (medial to lateral), mm	39.0 [37; 41]	39.0 [37;41]	>0.05
LA diameter (front to back), mm	53.0 [49 ;54]	52.8 [49; 55]	>0.05
RA diameter (medial to lateral), m	36.6 [35 ;39]	36.5 [34; 39]	>0.05
RA diameter (front to back), mm	49.0 [47; 51]	48.2 [45; 50]	>0.05
LV ESD, mm	35.2 [32; 38]	36.5 [33; 38]	>0.05
LV EDD, mm	51.1 [49; 53]	50.5 [47; 52]	>0.05
M-mode	-	-	-
LV ESV, ml	53.9 [42.5; 63]	62.3 [46; 62]	0.004
LV EDV, ml	130.0 [113; 141]	131.5 [104; 138]	>0.05
LVEF, %	58.9 [53; 64]	53.6 [52; 60]	0.001
B-mode	-	-	-
LV ESV, ml	58.8 [48; 65]	60.7 [46.5; 70]	>0.05
LV EDV, ml	128.6 [110; 140]	120.4 [100; 137]	>0.05
LVEF, %	54.0 2[51; 59]	49.9 [45; 54.5]	0.001
Septal thickness (systolic), mm	16.9 [15; 18]	16.6 [16; 18]	>0.05
Septal thickness (diastolic), mm	12.8 [11.75; 14]	12.8 [12; 14]	>0.05
Posterior wall thickness (systolic), mm	15.6 [14; 17]	15.3 [14; 17]	>0.05
Posterior wall thickness (diastolic), mm	11.2 [10; 12]	11.3 [10; 12]	>0.05
Contractility index	1.27 [1.06;1.395]	1.49 [1.19; 1.75]	0.001
Right ventricle diameter, mm	26.5 [24.5%; 28]	25.9 [24; 28]	>0.05
TAPSE, mm	18.5 [17; 20]	19.6 [19; 22]	>0.05
MR grade 1, N (%)	31 (48.4)	10 (40)	>0.05
MR grade 2, N (%)	18 (28.1)	7 (28)	>0.05
MR grade 3, N (%)	5 (7.8)	1 (4)	>0.05
TR grade 1, N (%)	5 (7.8)	10 (40)	>0.05
TR grade 2, N (%)	13 (20.3)	14 (56)	>0.05
TR grade 3, N (%)	1(1.5)	-	>0.05
AR grade 1, N (%)	15 (23.4)	10 (40)	>0.05
AR grade 2, N (%)	6 (9.3)	-	>0.05

Abbreviations: LA, left atrium; RA, right atrium; LV, left ventricle; ESD, end-systolic diameter; EDD, end-diastolic diameter; ESV, end-systolic volume; EDV, end-diastolic volume; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; TR, tricuspid regurgitation

According to the results of transthoracic echocardiography, patients in both groups were comparable in terms of the diameters of both the atria and ventricles. However, patients of Group 2 had larger end-systolic volume of the left ventricle (62 [46; 62] vs 54 [42.5; 63] ml p=0.004), and lower LVEF (49 [45; 54] % vs 54 [51; 59] %, p=0.001), suggesting more extensive left ventricular necrosis and ischemia, which may contribute to the development of ventricular tachycardia or fibrillation. However, other linear and volumetric parameters did not show significant intergroup differences (p>0.05).

DISCUSSION

The clinical, laboratory, and echocardiographic characteristics of patients with ST elevation MI complicated by ventricular arrhythmias (Group 2) were the primary focus of this study. We identified several distinct

parameters that were more prominent in these patients than in those with uncomplicated STEMI.

The hospital mortality rate for patients with STEMI and malignant VAs is greater than that for patients with STEMI alone. Several underlying mechanisms exist. Main coronary artery narrowing causes a major mismatch between myocardial perfusion and demand, leading to myocyte death from ischemia and electrical alterations in the ventricular myocardium. If left untreated, this might lead to electrical instability, which can show up as VT or VF and cause abrupt cardiac death.[9]

During the ischemic phase of myocardial infarction (MI), mechanisms such as automaticity, triggered activity, and reentry play a major role in inducing arrhythmias.[10] In the acute phase of myocardial ischemia, dysregulation of potassium (K+), sodium (Na+), and calcium (Ca2+) ion channels, along with reduced intercellular electrical coupling, promotes conduction heterogeneity. This heterogeneity leads to impaired conduction of impulses. Furthermore, fibrotic connective tissue and the marginal zone display the nature of poor electrical coupling, slow conduction of electrical signals, unidirectional block, and reentry conduction, which can start sustained ventricular tachycardia (VT).[11]

Ventricular arrhythmias (VAs) are often connected with inferior myocardial infarction (MI), which generally influences the inferior part of the left ventricle (LV). The right coronary artery (RCA), which provides blood to the inferior wall of the LV and the atrioventricular (AV) node, participates a crucial role. Obstruction of the RCA can impair these regions, possibly leading to heart block due to AV node dysfunction, thereby affecting arrhythmogenic consequence in inferior MI. In addition, damaging of the inferior wall post-MI impairs the normal flow of electrical impulses, thereby elevating the risk of re-entry arrhythmias. The ischemic myocardium can also trigger potentially fatal VAs due to electrical instability following infarction.[12,13]

Increased CRP and WBC counts, elevated glucose levels, and noticeably higher troponin concentrations indicate more severe systemic inflammation and cardiac damage. In the context of STEMI, elevated WBC counts indicate an immediate inflammatory response that is linked to an increased risk of arrhythmia. Electrical instability can result from the modulation of ion channel expression and function by inflammation. Adverse outcomes have also been associated with metabolic and renal dysfunction markers, such as hyperglycemia and increased CRP, which may exacerbate cardiac stress and encourage arrhythmogenesis.[14-16]

An increase in troponin levels is a strong indicator of myocardial necrosis. According to previous studies displaying the infarct size is an important indicator of arrhythmic risk and mortality following STEMI, higher troponin levels in the VA group in this cohort revealed an increased degree of myocardial injury.[17,18]

Patients with ventricular arrhythmias (VAs) display larger end-systolic volumes and markedly lower left ventricular ejection fraction (LVEF) than patients without arrhythmias, as displayed by echocardiography. These results indicate more extensive left ventricular dysfunction and necrosis, which are proven predictors of early and late post-infarction arrhythmias. Decreased systolic function measured by LVEF, is mainly important as it reflects the degree of contractile abnormality and the possibility of adverse remodeling, both generating the arrhythmogenic foundation of this concomitant disease.[19,20]

Risk evaluation and treatment will be specially influenced by the discovery of clinical, laboratory, and echocardiographic variables of VAs in patients with STEMI. Furthermore, the control and early management may be crucial for patients with these high-risk features, especially those with elevated inflammation, metabolic disturbances, decreased ventricular function, and myocardial injury. This could result in aggressive management of metabolic abnormalities, timely revascularization, and in certain conditions, the assessment of preventative antiarrhythmic controls.

A considerable fraction of post-myocardial infarction (MI) patients tends to be at increased risk for sudden cardiac death as a consequence of prolonged VAs, particularly in the early post-infarction phase, in spite of advancements in reperfusion therapy and pharmaceutical management. Therefore, elevating the outcomes requires the early identification and focused management of high-risk category patients.

CONCLUSION

Patients with STEMI followed by ventricular tachyarrhythmias displayed several key differences in comparison with patients without Ventricular Arrythmia (VA), underscoring the significant factors that may lead to arrhythmia development.

These patients had a markedly higher prevalence of inferior MI and displayed increased WBCs and glucose levels, both of which are suggestive of intrinsic inflammation and metabolic abnormalities. Furthermore, troponin levels were significantly higher in the VA group, supporting the concept that myocardial injury and damage are predisposing factors. The lower left ventricular ejection fraction and the elevated contractility index in these patients indicate more widespread myocardial injury, which is a known predecessor to the growth of ventricular tachycardia and fibrillation.

Collectively, these findings emphasize that inflammatory markers, myocardial injury, impaired ventricular function, and metabolic disturbances play a significant role in the pathophysiology of arrhythmias in patients with STEMI,

highlighting the importance of early monitoring and intervention in these patients to mitigate arrhythmic risks.

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