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Original Research Article

Outcome analysis in patients with non-ST elevation acute coronary syndrome

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ABSTRACT

Background: Non-ST-elevation acute coronary syndrome (NSTE-ACS) represents a heterogeneous clinical entity ranging from unstable angina to non-ST-elevation myocardial infarction (NSTEMI). Despite similar initial clinical presentation, patient outcomes vary considerably. Early identification of factors associated with progression to MI remains clinically important for risk stratification and management optimization. Aim was to compare clinical, laboratory, and echocardiographic characteristics of patients presenting with NSTE-ACS according to in-hospital diagnosis.
Methods: A prospective observational study included 98 patients admitted with NSTE-ACS to the Grodno Regional Cardiological Center (Belarus). Patients were divided into two groups: Group 1-MI (n=51) and Group 2-unstable angina (n=47). All patients underwent clinical evaluation, laboratory testing including cardiac biomarkers, and coronary angiography with possible percutaneous coronary intervention. Statistical analysis was performed using non-parametric methods with significance set at $p<0.05$.
Results: Baseline demographic characteristics were comparable between groups. Patients with MI demonstrated significantly higher leukocyte counts ($p=0.005$), higher creatinine levels ($p=0.01$), and significantly different total cholesterol values ($p=0.0003$). Echocardiographically, patients with unstable angina showed larger left atrial dimensions ($p=0.017$ and $p=0.031$) and greater left ventricular (LV) end-diastolic diameter ($p=0.033$). No significant differences were observed in LV ejection fraction.
Conclusions: Patients with NSTE-ACS who develop MI exhibit more pronounced inflammatory activation, whereas those with unstable angina demonstrate more prominent structural cardiac remodeling. These findings suggest that both inflammatory burden and cardiac structural characteristics may contribute differently to clinical outcomes in NSTE-ACS.

Keywords: Non-ST-elevation acute coronary syndrome, NSTEMI, Unstable angina, Echocardiography, Inflammation, Cardiac remodeling

INTRODUCTION

Cardiovascular disease remains the leading cause of mortality worldwide, accounting for nearly one-third of all global deaths, as reported by the World Health Organization.[1] Among cardiovascular conditions, ACS represents a major contributor to hospital admissions and long-term morbidity. Despite advances in prevention and revascularization strategies, the burden of ischemic heart disease continues to grow, particularly in aging populations with multiple comorbidities.

NSTE-ACS includes unstable angina and NSTEMI. Both entities arise from disruption of an atherosclerotic plaque

followed by platelet aggregation and thrombus formation, resulting in partial or transient coronary obstruction.[2,3] The key distinguishing feature is myocardial necrosis, confirmed by elevation of cardiac troponins in NSTEMI, while unstable angina occurs without detectable cardiomyocyte death according to the Fourth Universal Definition of Myocardial Infarction.[4]

The introduction of high-sensitivity troponin assays has substantially improved early detection of myocardial injury.[5] However, troponin elevation represents the final step of myocardial damage rather than a predictor of its development. Contemporary guidelines from the European Society of Cardiology and the American Heart Association[7] emphasize the importance of early risk stratification using clinical presentation, ECG changes, biomarkers, and validated risk scores such as GRACE.[6,8]

Inflammation plays a central role in atherosclerotic plaque instability. The inflammatory hypothesis of atherosclerosis, extensively described by Libby and colleagues, proposes that immune cell activation weakens the fibrous cap and promotes plaque rupture.[9] Elevated leukocyte count and C-reactive protein levels have been associated with worse outcomes in ACS.[10,11]

Renal dysfunction is another recognized predictor of adverse outcomes in ACS. Even mild reductions in glomerular filtration rate are associated with higher mortality and recurrent ischemic events.[12,13] Pathophysiological mechanisms include endothelial dysfunction, oxidative stress, chronic inflammation, and altered platelet reactivity.[12]

Beyond biochemical markers, cardiac imaging plays a central role in the assessment of patients with NSTE-ACS. Transthoracic echocardiography remains a widely available and non-invasive modality that provides essential information regarding global and regional ventricular function, chamber dimensions, wall thickness, and hemodynamic status.[14] While LV ejection fraction (LVEF) is traditionally regarded as a major prognostic indicator, increasing evidence suggests that structural parameters-such as left atrial (LA) enlargement and LV remodeling-may also carry important clinical implications.[16]

Echocardiography provides essential structural and functional information in ACS patients. Beyond assessing LV ejection fraction, echocardiography enables evaluation of chamber size, wall thickness, and ventricular remodeling.[14] Left atrial enlargement is associated with chronic diastolic dysfunction and long-standing pressure overload.[15] Similarly, increased LV end-diastolic diameter and volume may reflect chronic ischemic remodeling rather than acute necrosis.

While large registries such as GRACE have provided important epidemiological insights, few studies directly compare laboratory and echocardiographic differences between NSTEMI and unstable angina within the same cohort.[8] Understanding these distinctions may help clarify pathophysiological mechanisms underlying progression to MI.

In this context, we conducted a prospective observational study to evaluate clinical, laboratory, and echocardiographic characteristics of patients presenting with NSTE-ACS and to compare findings between those diagnosed with NSTEMI and unstable angina. We hypothesized that inflammatory activation and renal impairment would be more pronounced in NSTEMI, whereas structural cardiac remodeling would be more evident in unstable angina.

Aim

The study was to compare clinical, laboratory, and echocardiographic characteristics of patients presenting with NSTE-ACS according to in-hospital diagnosis (NSTEMI versus unstable angina), and to identify parameters associated with MI development.

METHODS

The study included 98 patients with NSTE-ACS who were treated at the Grodno Regional Clinical Cardiological Center (Belarus) in 2025. Patients were divided into 2 categories according to their outcomes: Group 1 included 51 patients with MI, and Group 2 included 47 patients with unstable angina.

Exclusion criteria from the study were: chronic rheumatic heart disease, acute myocarditis, pericarditis or endocarditis, pulmonary embolism, acute aortic dissection, valvular pathology of the heart requiring surgical correction, and severe concomitant extracardiac pathology.

In this study, patients were diagnosed with MI based on clinical symptoms, signs, ECG findings, and cardiac biomarker values.

Patients underwent coronary angiography with possible percutaneous coronary intervention (PCI) at the same center according to the Judkins method (1967) in the X-ray operating room on the 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 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, the distribution of which was not normal, were given as a median, 25% and 75% quartiles. Since most of the quantitative characteristics did not obey the normal distribution law, non-parametric methods were used for comparison. The Mann-Whitney 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 prior to inclusion in study.

RESULTS

Clinical and anamnestic characteristics of the studied patients are presented in Table 1.

Table 1 summarizes the baseline clinical characteristics of patients with NSTE-ACS, stratified into Group 1 (n=51) and Group 2 (n=47), with p values for intergroup comparisons (likely chi-square for categorical variables and Mann-Whitney U for continuous variables).

The two groups were largely comparable in terms of demographic and major cardiovascular risk factors. No significant differences were observed in male gender predominance (62.7% in Group 1 vs. 70.2% in Group 2; p>0.05), median age (65 years [58; 72] vs. 67 years [63; 72]; p>0.05), body mass index (28.5 kg/m² [25.7; 32.4] vs. 30 kg/m² [26.8; 33.2]; p>0.05), prevalence of obesity (35.2% vs. 47%; p>0.05), diabetes mellitus (31.3% vs. 19.6%; p>0.05), or anemia (13.7% vs. 27.4%; p>0.05). Similarly, functional status as assessed by NYHA heart failure classification showed balanced distribution across classes 1-3 (Class 1: 31.3% vs. 29.4%; Class 2: 47% vs. 47%; Class 3: 21.5% vs. 23.5%; all p>0.05), with no patients in Class 4 in either group.

The only statistically significant difference (p=0.007) was a higher prevalence of paroxysmal atrial fibrillation (AF) in Group 2 (37.3%) compared to Group 1 (15.7%). Persistent AF (3.9% vs. 1.9%; p>0.05) and atrial flutter (0% vs. 1.9%; p>0.05) were infrequent and showed no significant intergroup difference.

Table 2 describes the localization of MI and in-hospital complications among patients in Group 1 (n=51), presented as absolute numbers and percentages.

The most frequent MI localization was anterior, affecting 20 patients (39.2%), followed by inferior in 17 patients (33.3%). Combined anterior-lateral involvement occurred in 12 patients (23.5%), while isolated lateral wall MI was the least common, seen in 6 patients (11.8%). These proportions indicate that anterior and inferior territories predominated in this cohort, accounting for over 70% of cases when considered together (with some overlap in anterior-lateral cases). This distribution is consistent with typical patterns in ACSs, particularly in cohorts enriched for ST-elevation MI (STEMI), where proximal left anterior descending (LAD) or right coronary artery (RCA) occlusions commonly produce anterior or inferior infarcts, respectively. Lateral wall involvement, often linked to left circumflex occlusion, was less prevalent, aligning with its lower overall incidence in MI series.

In-hospital complications were infrequent. Ventricular tachycardia developed in 2 patients (3.9%), pulmonary edema in another 2 patients (3.9%), and ventricular fibrillation was absent (0%).

Table 3 presents a comparative analysis of laboratory parameters between Group 1 (n=51) and Group 2 (n=47), reported as medians with interquartile ranges [25%; 75%], with p-values derived from non-parametric testing (Mann-Whitney U test).

Patients in Group 1 demonstrated significantly higher white blood cell counts (WBC) than those in Group 2 (median 9.4 × 10⁹/l [7.6; 11.1] vs. 7.8 × 10⁹/l [6.1; 9.2]; p=0.005).

Serum creatinine was significantly lower in Group 1 (median 89.37 µmol/l [72.0; 101.9] vs. 99.6 µmol/l [86; 114.35]; p=0.01), suggesting relatively preserved renal function at presentation in this group. Although the estimated glomerular filtration rate (eGFR) appeared numerically higher in Group 1 (median 97.65 mL/min/1.73 m² [60.05; 92.3] vs. 69.31 mL/min/1.73 m² [58.38; 82.15]), this difference did not achieve statistical significance (p>0.05).

A highly significant difference was noted in total cholesterol levels, with Group 1 exhibiting lower values (median 5.57 mmol/l [4.08; 7.03] vs. 6.74 mmol/l [3.2; 5.36]; p=0.0003).

No significant differences (all p>0.05) were observed for other parameters, including red blood cell count, hemoglobin, erythrocyte sedimentation rate, platelets, urea, LDL cholesterol, HDL cholesterol, triglycerides, glucose, liver enzymes (AST, ALT), electrolytes (sodium, potassium), NT-proBNP, D-dimers.

Markedly higher troponin values in Group 1 in comparison with Group 2 (p<0.001) strongly suggest greater myocardial necrosis in Group 1.

Table 4 presents a comparative analysis of echocardiographic parameters between Group 1 (n=51) and Group 2 (n=47), expressed as medians with interquartile ranges [25%; 75%], with p-values indicating statistically significant differences (p<0.05) in select structural indices.

Patients in Group 2 exhibited significantly larger left atrial (LA) dimensions in multiple planes compared to Group 1. Specifically, the LA diameter in the 2-chamber view was greater in Group 2 (median 40.14 mm [36; 42] vs. 37.6 mm [35; 39]; p=0.017), as was the front-to-back LA diameter (55.34 mm [50.5; 59] vs. 52.54 mm [48; 57]; p=0.031). These findings indicate more pronounced LA enlargement in Group 2, which is a recognized marker of chronic

pressure overload, diastolic dysfunction, or higher burden of comorbidities (e.g., hypertension, atrial fibrillation risk) in ACS settings.

LV dimensions also differed significantly, with Group 2 showing larger LV end-diastolic diameter (LV EDD: 53.16 mm [47; 57.5] vs. 49.25 mm [46; 52]; p=0.033) and greater B-mode LV end-diastolic volume (LV EDV: 145.3 ml [101.25; 172] vs. 112.26 ml [89; 129.75]; p=0.008). This suggests more extensive LV dilation in group 2, potentially reflecting greater adverse remodeling, prior ischemic burden/ attenuated compensatory mechanisms following index event.

Additional significant differences included mildly greater systolic septal thickness in Group 2 (17.23 mm [15.25; 19] vs. 16.53 mm [15; 18]; p=0.047), which may indicate a hypertrophic response to chronic pressure loading or measurement variability, though diastolic septal thickness showed no difference (p>0.05). No significant intergroup differences were found for LV ejection fraction (LVEF) in M-mode (≈59-60%) or B-mode (≈52-53%), LV end-systolic volumes, posterior wall thickness (systolic or diastolic), right atrial (RA) dimensions (except one borderline front-to-back plane at p=0.031), right ventricular diameter, TAPSE, contractility index, or the prevalence of pericardial/pleural effusions (all p>0.05).

Table 1: Clinical characteristics of patients.

Parameters	Group 1, (n=51)	Group 2, (n=47)	P value
Male gender	32 (62.7%)	33 (70.2%)	>0.05
Age (in years)	65 [58; 72]	67 [63; 72]	>0.05
Body mass index (kg/m^2)	28.5 [25.7; 32.4]	30 [26.8; 33.2]	>0.05
Obesity	18 (35.2%)	24 (47%)	>0.05
Diabetes mellitus	16 (31.3%)	10 (19.6%)	>0.05
Paroxysmal AF	8 (15.7%)	19 (37.3%)	0.007
Persistent AF	2 (3.9%)	1 (1.9%)	>0.05
Atrial flutter	-	1 (1.9%)	>0.05
Anemia	7 (13.7%)	14 (27.4%)	>0.05
Heart failure NYHA class			
Class 1	16 (31.3%)	15 (29.4%)	>0.05
Class 2	24 (47%)	24 (47%)	>0.05
Class 3	11(21.5%)	12 (23.5%)	>0.05

Table 2: Localization of MI and in-hospital complications.

Parameters	Group 1, (n=51)
MI localization	
Anterior	20 (39.2%)
Inferior	17 (33.3%)
Lateral	6 (11.7%)
anterior-lateral	12 (23.5%)
Complications	
Ventricular tachycardia	2 (3.9%)
Pulmonary edema	2 (3.9%)
Ventricular fibrillation	0

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

Parameters	Group 1, (n=51)	Group 2 (n=47)	P value
RBC, (10^{12}/l)	4.5 [4.2; 4.9]	4.4 [4.0; 4.9]	>0.05
Hemoglobin, (g/l)	137 [127.7; 152]	133 [125;148]	>0.05
WBC, (10^9/l)	9.4 [7.6; 11.1]	7.8 [6.1; 9.2]	0.005
ESR, (mm/h)	14.86 [6; 18.7]	18.1 [8; 22]	>0.05
Platelets, (10^9/l)	245.94 [197.2; 297.2]	238.87 [197; 283]	>0.05
Urea, (mmol/l)	6.85 [5.15;7.5]	6.78 [4.65;8.1]	>0.05
Creatinine, (µmol/l)	89.37 [72.0;101.9]	99.6 [86;114.35]	0.01
eGFR, (ml/min/1.73 m^2)	97.65 [60.05;92.3]	69.31 [58.38;82.15]	>0.05
Total cholesterol, (mmol/l)	5.57 [4.08;7.03]	6.74 [3.2;5.36]	0.0003
LDL, (mmol/l)	3.9 [1.47;3.35]	2.14 [1.63;2.38]	>0.05
HDL, (mmol/l)	1.13 [0.78;1.23]	1.11 [0.93;1.33]	>0.05
Triglycerides, (g/l)	1.64 [1.05;2.03]	1.68 [1.18;2.1]	>0.05

Continued.

Parameters	Group 1, (n=51)	Group 2 (n=47)	P value
Glucose, (mmol/l)	7.93 [5.72;9.63]	6.68 [5.18;7.42]	>0.05
AST, (IU/l)	36.49 [19.4;36.93]	36.48 [15.48;31.75]	>0.05
ALT, (IU/l)	30.46 [15.85;35]	33.14 [17;42.6]	>0.05
Sodium, (mEq/l)	138.34 [137;140]	140.02 [138;143]	>0.05
Potassium, (mEq/l)	4.40 [4;4.75]	4.51 [4.1;4.7]	>0.05
NT-proBNP, (pg/ml)	5418 [3259.25;8182.5]	6818.91 [525.5;10253.5]	>0.05
D-dimers, (pg/ml)	2962.56 [731;3986]	2983.22 [1248;5000]	>0.05
Troponin (initial value), (ng/l)	394 [120.25;2303.25]	12 [4.75;59.2]	<0.001
Troponin (1 hour), (ng/l)	4305.18 [509;2902]	15.5 [3.98;137]	<0.001

*RBC-red blood cells; WBC-white blood cells; ESR-erythrocyte sedimentation rate; eGFR-estimated glomerular filtration rate; LDL-low-density lipoproteins; HDL-high-density lipoproteins; AST-aspartate aminotransferase; ALT-alanine aminotransferase.

Table 4: Echocardiographic parameters of patients (Me [25%;75%]).

Variables	Group 1, (n=51)	Group 2, (n=47)	P value
Parameters			
LA diameter (2 chamber), (mm)	37.6 [35;39]	40.14 [36;42]	0.017
LA diameter (medial to lateral), (mm)	39.21 [36.75;41]	40.66 [37.25;44]	>0.05
LA diameter (front to back), (mm)	52.54 [48;57]	55.34 [50.5;59]	0.031
RA diameter (medial to lateral), (mm)	37.20 [34;38]	37.29 [34;41.75]	>0.05
RA diameter (front to back), (mm)	50.06 [47;52]	50.76 [46.25;54]	>0.05
LV ESD, (mm)	34.04 [31.75;36.25]	37.71 [32;41]	>0.05
LV EDD, (mm)	49.25 [46;52]	53.16 [47;57.5]	0.033
M-mode			
LV ESV, (ml)	50.79 [40.75;55.5]	71.86 [43.75;80.25]	>0.05
LV EDV, (ml)	119.25 [110.25;132.5]	146.89 [109.5;157.75]	>0.05
LVEF, (%)	59.71 [56.5;64]	54.57 [51;61.5]	>0.05
B-mode			
LV ESV, (ml)	50.81 [37.25;66.5]	76.43 [38.75;101.75]	>0.05
LV EDV, (ml)	112.26 [89;129.75]	145.3 [101.25;172]	0.008
LVEF, (%)	52.69 [48;60]	52.32 [43.5;62.5]	>0.05
Septal thickness (systolic), (mm)	16.53 [15;18]	17.23 [15.25;19]	0.047
Septal thickness (diastolic), (mm)	12.71 [11;14]	13.39 [12;15]	>0.05
Posterior wall thickness (systolic), (mm)	15.9 [15;17]	16.5 [15;17.75]	>0.05
Posterior wall thickness (diastolic), (mm)	11.51 [10;12]	12 [11;13]	>0.05
Right ventricle diameter, (mm)	26.56 [24;27]	25.58 [24;28]	>0.05
TAPSE, (mm)	16.79 [16;19.5]	16.43 [13;19]	>0.05
Contractility index	1.36 [1;1.56]	1.23 [1;1.49]	>0.05
Pericardial effusion	10 (19.6%)	4 (8.51%)	>0.05
Pleural effusion	8 (15.69%)	4 (8.51%)	>0.05

*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; TAPSE-tricuspid annular plane systolic excursion.

DISCUSSION

In this observational study of patients admitted with NSTE-ACS, we aimed to better understand the clinical, laboratory, and echocardiographic differences between those who ultimately developed MI and those diagnosed with unstable angina. One of the most striking findings in our study was the significantly higher leukocyte count in patients with MI. This observation reinforces the central role of inflammation in plaque destabilization and myocardial injury.[11] The inflammatory paradigm of atherosclerosis, described by Peter Libby, proposes that immune activation weakens the fibrous cap and promotes rupture. In the setting of the NSTE-ACS, an elevated leukocyte count may therefore reflect not only systemic inflammatory burden but also local coronary plaque activity.[2]

Interestingly, serum creatinine levels differed significantly between groups. Although renal dysfunction is traditionally associated with worse cardiovascular outcomes, our data showed lower creatinine levels in the MI group at presentation.[13]

This somewhat paradoxical finding may reflect multiple factors, including earlier hospital presentation, differences in baseline comorbidity burden, or hemodynamic variations.

Another unexpected but highly significant result was the difference in total cholesterol levels between groups. While hyperlipidemia is a well-established risk factor for coronary artery disease, acute coronary events are sometimes associated with transient reductions in lipid levels due to inflammatory and metabolic shifts. This phenomenon may partially explain our findings.

From an echocardiographic standpoint, patients with unstable angina demonstrated larger LA dimensions and greater LV end-diastolic diameters and volumes. These findings suggest more pronounced structural remodeling in this group.[16] LA enlargement is a recognized marker of chronic diastolic dysfunction. It has been shown to predict adverse cardiovascular outcomes and reflects long-standing exposure to pressure or volume overload.[15]

In contrast, LVEF did not differ significantly between groups. This observation is clinically relevant. It suggests that preserved systolic function does not exclude substantial ischemic or structural pathology

The pattern that emerges from our results is one in which MI appears more closely associated with acute inflammatory activation, while unstable angina is characterized by more chronic structural cardiac adaptation. These processes are not mutually exclusive but may represent different stages or phenotypes within the spectrum of NSTE-ACS. Understanding these phenotypic differences may help refine risk stratification beyond troponin elevation alone.

This study deepened our appreciation for the multidimensional nature of ACSs. NSTE-ACS is not a uniform diagnosis; rather, it is a dynamic clinical state in which inflammation, thrombosis, myocardial oxygen supply-demand mismatch, and structural remodeling interact in complex ways. Recognizing this heterogeneity is essential if we aim to personalize treatment strategies.

Limitations

The main limitation of this study is the small sample size, from a single hospital. Additionally, other important factors that may play a role in these findings, including genetic polymorphisms and blood clotting factor levels, were not assessed. Nonetheless, our results must be interpreted with caution and larger studies with higher patient numbers should be carried out to confirm findings.

CONCLUSION

In conclusion, our study demonstrates that among patients presenting with NSTE-ACS, those who develop MI exhibit more pronounced inflammatory activation, as reflected by elevated leukocyte counts, while patients with unstable angina show more evident structural cardiac remodeling, particularly LA enlargement and increased LV dimensions. Further large-scale, multicenter studies are needed to validate our observations and determine their prognostic relevance over long-term follow-up. Incorporating high-sensitivity inflammatory biomarkers, advanced echocardiographic techniques and cardiac magnetic resonance imaging could clarify the relationship between inflammation, microvascular dysfunction, and remodeling. In addition, exploring genetic susceptibility, platelet function testing, and anti-inflammatory therapies may help identify patients at highest risk of progression from unstable angina to MI.

Future research should aim to construct risk models combining inflammatory markers, renal function parameters, structural cardiac indices, and clinical variables. Such an approach could ultimately lead to more tailored treatment strategies, improved prevention of myocardial injury, and better outcomes for patients worldwide.

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