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Original Research Article

Gender differences in patients presenting with non-ST elevation acute coronary syndromes

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ABSTRACT

Background: Although a role for gender-based differences in the development and associated complications of non-ST elevation acute coronary syndromes (NSTE-ACS) has been theorized, there is currently high uncertainty on the actual contribution of these factors in the epidemiology and clinical course of NSTE-ACS.
Methods: The retrospective study included 96 patients with NSTE-ASC, who were admitted to the Grodno Regional Clinical Cardiological Center (Belarus) for treatment over a period from January to October 2025. Patients were divided into 2 groups according to their gender: group 1 included 49 male patients, while group 2 – included 47 female patients. All patients underwent clinical, laboratory, and instrumental studies. Statistical analysis was performed using STATISTICA 12.0.
Results: Female patients were significantly older (p=0.005); and more often had obesity (p=0.02). Female patients were significantly more diagnosed with unstable angina (p<0.001), while in male patients myocardial infarction (MI) diagnosis was more common. High-sensitive troponin I levels were comparable between two groups (p=0.12), as well as lactate dehydrogenase (p=0.09) and creatine phosphokinase MB (p=0.98). The results of coronary angiography show a tendency towards more extensive multivessel coronary artery disease in men (35% versus 17%, p=0.048).
Conclusions: These findings emphasize the importance of gender-specific approaches to the diagnosis and risk stratification of ACS. Larger, multicentre prospective studies incorporating additional biomarkers and long-term outcome data are needed to validate these results and to advance a more balanced approach to the gender-specific management of ACS.

Keywords: Non-ST-elevation acute coronary syndrome, Myocardial infarction, Unstable angina, Troponin, Male gender, Female gender

INTRODUCTION

Acute coronary syndromes (ACS) remain a leading cause of morbidity and mortality worldwide. According to the American College of Cardiology, ACS generally results from the rupture of an unstable coronary artery atherosclerotic plaque, which leads to partial or complete blockage by a blood clot and/or microemboli. This blockage reduces blood flow to the heart muscle, causing myocardial ischemia. ACS includes three related conditions on a severity spectrum: unstable angina, non-ST elevation myocardial infarction (NSTEMI), and ST elevation myocardial infarction (STEMI). The initial diagnosis and classification should be based on clinical symptoms, ECG interpretation, and cardiac troponin (cTn) levels. Among these, non-ST elevation ACS (NSTE-ACS), which includes NSTEMI and unstable angina, makes up most ACS hospital admissions and presents a unique diagnostic challenge due to often non-specific symptoms and ECG changes.[1]

In recent decades, interest in the issue of gender differences in cardiovascular risk factors has grown significantly. Women of childbearing age, unlike men, have a low risk of cardiovascular events, and the period of increase in cardiovascular disease incidence is observed among women 10 years later than among men and is associated with the onset of the perimenopausal period.[2] Coronary artery atherosclerosis progresses differently in men and women: men develop stenotic coronary lesions earlier and more frequently, while women are more likely to develop non-stenotic coronary atherosclerosis or microvascular dysplasia and endothelial dysfunction.[2]

Understanding these gender differences is vital for enhancing patient outcomes across the entire healthcare process. Ongoing attention to gender disparities helps improve understanding, as analyzing gender-related data can lead to earlier, more precise diagnoses, tailored treatments, and better prognosis and prevention strategies.

Therefore, aim of the study was to identify clinical, laboratory and angiographic differences in male and female patients presenting with NSTE-ACS.

METHODS

The single-center retrospective study included 96 patients with NSTE-ASC, who were admitted to the Grodno Regional Clinical Cardiological Center (Belarus) for treatment over a period from January 2025 to October 2025. NSTE-ASC was diagnosed as anginal chest pain and new or presumed new horizontal or down-sloping ST-segment depression ≥0.5 mm in ≥2 contiguous ECG leads and/or T-wave inversion >1 mm in ≥2 contiguous leads with prominent R wave or R/S ratio >1 or transient ST-segment elevation. According to our further protocol, a definite diagnosis of NSTE-ACS was given to those patients with MI (high-sensitive troponin I/T elevation >99 percentile at presentation (0 hours) or 1 hour later or unstable angina (without troponin elevation but with evidence of ischemia on ECG). Patients were divided into 2 groups according to their gender: group 1 included 49 male patients (51%), while group 2 – included 47 female patients (49%).

Exclusion criteria from the study were: STEMI; high-sensitive troponin I/T elevation due to myocarditis, pericarditis, infective endocarditis, pulmonary embolism, acute aortic dissection and other cardiac and/or extracardiac pathology.

In this study, patients underwent clinical and laboratory investigation, including a high-sensitive troponin I/T test upon admission and/or in 1 hour. Coronary angiography with possible percutaneous coronary intervention (PCI) 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 $p<0.05$, 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.

RESULTS

Clinical and anamnestic characteristics of the studied patients are presented in Table 1. Significant gender related variations were observed in several laboratory indices (Table 2). The results of coronary angiography also show a tendency towards more extensive coronary artery disease in men (34.69% in men versus 17.02% in women; p=0.0486) (Table 3).

Table 1: Clinical characteristics of patients.

Parameters	Group 1 (n=49)	Group 2 (n=47)	P value
Age, years	61.4 [56; 67]	66.96 [60; 75]	0.006
Body mass index, kg/m^2	27.83 [25.78; 30.55]	31.07 [27.4; 33.8]	0.029
Obesity, N (%)	9 (18.37)	19 (40.43)	0.018
Hypertension, N (%)	42 (85.71)	42 (89.36)	>0.05
Stage 1, N (%)	6 (12.24)	5 (10.64)	>0.05
Stage 2, N (%)	34 (69.39)	32 (68.09)	>0.05
Stage 3, N (%)	2 (4.08)	5 (10.64)	>0.05
Previous MI, N (%)	7 (14.29)	12 (25.53)	>0.05
Diabetes mellitus, N (%)	8 (16.33)	14 (29.79)	>0.05
Anemia, N (%)	4 (8.16)	9 (19.15)	>0.05
AF paroxysmal, N (%)	5 (10.20)	3 (6.38)	>0.05
AF persistent, N (%)	4 (8.16)	4 (8.51)	>0.05

Continued.

Parameters	Group 1 (n=49)	Group 2 (n=47)	P value
NYHA HF class	1.86 [1; 3]	1.95 [1; 2.25]	>0.05
MI localization, N (%)			
Anterior	12 (24.49)	12 (25.53)	>0.05
Inferior	21 (42.86)	14 (29.79)	>0.05
Lateral	11 (22.45)	5 (10.64)	>0.05
Anterior-lateral	5 (10.20)	7 (14.89)	>0.05
MI complications, N (%)			
Ventricular tachycardia	6 (12.24)	11 (23.4)	>0.05
Pulmonary edema	6 (12.24)	14 (29.79)	0.034
Cardiogenic shock	6 (6.12)	0 (0)	0.013
Ventricular fibrillation	1 (2.04)	0 (0)	>0.05

MI – Myocardial infarction; AF – atrial fibrillation; NYHA – New York Heart Association

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

Parameters	Group 1 (n=49)	Group 2 (n=47)	P value
RBC, 10^{12}/l	4.72 [4.44; 5.05]	4.34 [4.14; 4.69]	0.003
Hemoglobin, g/l	145 [138.25; 152.5]	131 [124.5; 141.5]	0.0001
WBC, 10^9/l	9.49 [7.38; 10.98]	9.62 [7.55; 11.65]	>0.05
ESR, mm/hour	13.88 [5; 15.5]	19.78 [10.5; 24.75]	0.003
Platelets, 10^9/l	252.54 [185; 261]	254.65 [185; 315]	>0.05
Urea, mmol/l	6.72 [5.18;7.15]	6.78 [4.8; 7.75]	>0.05
Creatinine, µmol/l	94.83 [77.9; 103.25]	85.45 [64.63; 100.65]	0.014
eGFR, ml/min/1.73 m^2	83.57 [68.5; 97.63]	71.83 [58.55; 87.58]	0.019
Total cholesterol, mmol/l	5.59 [4.41; 6.49]	5.55 [3.91; 6.84]	>0.05
Glucose, mmol/l	8.19 [6.1;8.8]	9.98 [5.9; 10.93]	>0.05
LDL, mmol/l	2.79 [1.96; 3.45]	2.85 [2.1; 3.2]	>0.05
HDL, mmol/l	1.04 [0.82;1.18]	1.18 [0.91; 1.46]	>0.05
Triglycerides, g/l	1.84 [1.16; 2.8]	1.67 [1.17; 2.13]	>0.05
AST, IU/l	38.5 [21.5;46]	45.27 [21; 56.15]	>0.05
ALT, IU/l	32.27 [19.8; 37.5]	34.53 [20; 34.75]	>0.05
Sodium, mEq/l	139.33 [135; 142]	138.97 [136.4; 140]	>0.05
Potassium, mEq/l	4.33 [4; 4.60]	4.21 [3.9;4.6]	>0.05
Fibrinogen, g/l	5.00 [3.36; 5.97]	4.75 [3.36; 5.57]	>0.05
High-sensitive troponin I, ng/l	5291.42 [3.35; 4209.5]	4282.26 [127.5; 3769.5]	>0.05
Lactate dehydrogenase, IU/l	493.91 [350.5 ;549]	590.16 [409.25; 645]	>0.05
Creatine phosphokinase MB, IU/l	493.91 [20.03; 66.43]	74.47 [16; 73]	>0.05

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 3: Coronary angiography parameters.

Parameters	Group 1 (n=49)	Group 2 (n=47)	P value
Affected vessel, N (%)	-	-	-
Left main coronary artery	15 (1)	9 (19.14)	>0.05
Left anterior descending artery	42 (85.71)	33 (70.21)	>0.05
Left circumflex artery	29 (59.18)	26 (55.31)	>0.05
Right coronary artery	36 (73.46)	30 (63.82)	>0.05
Ramus intermedius	27 (55.10)	18 (38.29)	>0.05
Stenting	28 (57.14)	22 (46.80)	>0.05
Total number of stents	1.67 [1; 2]8	1.73 [1; 2]	>0.05
Number of vessel disease (stenosis >50%), N (%)			
1 Affected vessel	8 (16.32)	14 (29.78)	>0.05
2 Affected vessels	15 (30.61)	10 (21.2)	>0.05
3 or more affected vessels	17 (34.69)	8 (17.02)	0.049

DISCUSSION

Females presented at an older age compared to males (p=0.006), which is consistent with current literature which suggests that acute coronary syndromes occur in women approximately 5-10 years later than in men. It is primarily due to the protection provided by endogenous estrogen in the premenopausal period.[12] Several positive effects of estrogen on the cardiovascular system include maintenance of desirable lipid levels, improved endothelial function, reduced inflammation and direct vasodilation.[3] With reduction in estrogen following menopause, there is a rise in the risk of atherosclerosis, explaining the delayed presentation of disease in females.[4]

A higher body mass index was also observed in female patients (p=0.028) and also more obese patients (40.43% versus 18.37%; p=0.017). These findings reflect the findings made by Kinsara and Ismail who discovered that among NSTEMI patients, gender discrepancies were present with regard to metabolic risk factors.[1,5]

Women were nearly eight times more likely to be diagnosed with unstable angina, suggesting that female patients may have subtler or delayed troponin release patterns, smaller infarct sizes, or a different underlying pathophysiology that results in less myocardial necrosis.[7] Several mechanisms have been proposed regarding this. One of which is that women tend to suffer more frequently from non-obstructive coronary artery disease, also referred to as MINOCA. It makes up for 6-15% of all acute coronary syndrome presentations and affects 2-3 times as many women than men.[8,9] Patients suffering from MINOCA experience smaller or less sustained elevations in troponins, which may fall under the threshold required for diagnosis.[8,10]

A greater prevalence of pulmonary edema among women could be explained by the underlying pathophysiology of the disease. More female patients suffered from hypertension (89.36%), obesity (40.43%), and diabetes mellitus (29.79%), suggesting a stronger presence of risk factors associated with diastolic dysfunction.[12] Research has shown that female patients suffer from heart failure with preserved ejection fraction (HFpEF) more frequently than male patients, and such cases tend to develop rapidly during acute ischemia or hypotension.[13]

In contrast, the absence of cardiogenic shock in female patients, despite their older age and greater comorbidity burden, is also important. This finding can be explained by the significantly lower prevalence of multivessel coronary artery disease in women (three or more affected vessels: 17.02% versus 34.69%, p=0.048).[14] Cardiogenic shock is typically caused by ischemia or infarction affecting a great part of the left ventricular myocardium (over 40%).[15] Those who suffer from multiple coronary vessel disease are at a higher risk due to the simultaneous involvement of several vascular areas. Additionally, women have less left ventricular mass. Therefore, the volume of myocardium at risk in case of occlusion of coronary arteries is smaller and has a lower chance of pump failure.[16]

There was also a tendency towards more frequent ventricular tachycardia in men (23.4% in men versus 12.24% in women), which corresponds to greater scar burden and larger infarcts, which may be associated with multivessel disease.[17]

Female patients exhibited a lower number of erythrocytes (p=0.0029) and hemoglobin content (p=0.0002), accompanied by increased erythrocyte sedimentation rate (ESR) (p=0.003). While there was no statistically significant difference in the frequency of anemia (19.15% versus 8.16%, p=0.11), the consistent pattern of lower hemoglobin in women is clinically relevant.[11]

The complication of anemia associated with ACS reduces the oxygen carrying capacity of blood, aggravates myocardial ischemia, and is associated with increased mortality rates and major adverse cardiovascular events. It appears that the effect of anemia on the outcome of disease is more prominent in females, which could be due to their comparatively lower hemoglobin levels and physiological reserve.[11] Increased prevalence of pulmonary edema observed in women (29.8% compared to 12.2%, p=0.034) might stem from anemia as low oxygen supply may cause heart failure due to increased oxygen demand.[12]

Increased levels of ESR in women were suggestive of systemic inflammation. High levels of inflammatory markers are associated with poor prognosis in cases of ACS and may indicate a greater risk of plaque erosion, which involves less inflammation but elicits a better systemic inflammatory response than plaque rupture.[1]

This finding is consistent with previous large registries such as ACOS, according to which women with NSTE-ACS have more single-vessel disease and non-obstructive coronary artery disease.[2]

In both groups, the vessel most frequently affected was the left anterior descending artery (LAD), which was slightly more affected in men (85.71% versus 70.21%, p=0.0663). LAD artery supplies a broad territory including the front wall, the septum, and the apex. If this artery becomes occluded, this leads to the highest mortality rate compared with other single-vessel diseases.[15]

The phenomenon of women having less extensive CAD disease than men but elevated troponins similar to men can be explained by the recent research showing that ACS is caused more frequently by non-obstructive or microvascular causes in women. Coronary microvascular dysfunction (CMD) refers to the inability of microvessels in the heart to relax even without obstruction of the larger arteries. CMD is more prevalent in women and could result in ischemia, angina, and even myocardial infarction despite a normal or almost normal coronary arteries.[18,19]

CMD patients exhibit chest pain and electrocardiography abnormalities characteristic of ischemia, as well as elevated levels of troponins that tend to be less than in cases of heart diseases obstructing blood flow. In some cases, completely normal results of coronary angiography have also been observed.[18] The fact that women have a higher incidence of unstable angina (this category also encompasses troponin-negative acute coronary syndromes) and lower incidence of multi-vessel obstructive heart diseases fits well into the CMD paradigm.[19]

There were no statistically significant differences between male and female patients concerning the frequency of undergoing percutaneous coronary intervention with stenting (p=0.31) and the total number of implanted stents (p=0.55). These results demonstrate a similar accessibility to revascularization procedures among men and women, which contradicts certain previous reports suggesting less aggressive management of female patients.[20]

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, the results must be interpreted with caution and a study with a greater number of patients should be carried out to confirm these findings.

CONCLUSION

This study demonstrates significant gender-based differences in the clinical presentation, laboratory profile, and in-hospital course of patients with acute coronary syndromes. Female patients presented at a significantly older age and with a higher prevalence of obesity. Women were approximately eight times more likely to be diagnosed with unstable angina, accompanied by lower hemoglobin levels and elevated erythrocyte sedimentation rate, suggesting a greater contribution of non-obstructive mechanisms such as MINOCA and coronary microvascular dysfunction to ACS pathogenesis in females.

The significantly higher prevalence of pulmonary edema in women, due to greater diastolic dysfunction and reduced hemoglobin reserves, contrasts with the occurrence of cardiogenic shock only in male patients and a trend toward more frequent ventricular tachycardia in men. This is consistent with the more extensive multivessel obstructive coronary artery disease observed in this group. No significant disparity in revascularisation rates was identified between the two groups.

These findings emphasize the importance of gender-specific approaches to the diagnosis and risk stratification of ACS. The present study is limited by its single-centre design and small sample size. Larger, multicentre prospective studies incorporating additional biomarkers and long-term outcome data are needed to validate these results and to advance a more balanced approach to the management of acute coronary syndromes.

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