Sivkov O. G.
Surgut State University, Surgut, Khanty-Mansi Autonomous District-Ugra, Russia

# Factors Associated With Hospital Mortality in Acute Myocardial Infarction


| | |
|---|---|
| *Aim* | To determine clinical and laboratory parameters associated with in-hospital mortality in patients with acute myocardial infarction and to develop a multifactorial prognostic model of in-hospital mortality. |
| *Material and methods* | This was a study based on the 2019–2020 Registry of acute coronary syndrome of the Tyumen Cardiology Research Center, a branch of the Tomsk National Research Medical Center. The study included 477 patients with ST-segment elevation acute myocardial infarction (AMI), 617 patients with non-ST segment elevation AMI, and 26 patients with unspecified AMI. In-hospital mortality was 6.0% (n=67). Clinical and laboratory parameters were assessed on the day of admission. The separation power of indicators associated with in-hospital mortality was determined using a ROC analysis. The data array of each quantitative parameter was converted into a binary variable according to the obtained cut-off thresholds, followed by creation of a multifactorial model for predicting in-hospital mortality using a stepwise analysis with backward inclusion (Wald). The null hypothesis was rejected at $p<0.05$. |
| *Results* | The multivariate model for prediction of in-hospital mortality included age (cut-off, 72 years), OR 3.0 (95% CI: 1.5–5.6); modified shock index (cut-off threshold, 0.87), OR 1.5 (95% CI: 1.1–2.0); creatine phosphokinase-MB (cut-off threshold, 32.8 U/L), OR 4.1 (95% CI: 2.2–7.7); hemoglobin (121.5 g/l), OR 1.7 (95% CI: 1.2–2.3); leukocytes (11.5×109/l), OR 1.9 (95% CI: 1.3–2.6); glomerular filtration rate (60.9 ml/min), OR 1.7 (95% CI: 1.2–2.2); left ventricular ejection fraction (42.5%), OR 4.1 (95% CI: 2.0–8.3); and size of myocardial asynergy (32.5%), OR 2.6 (95% CI: 1.4–5.0). |
| *Conclusions* | Independent predictors of in-hospital mortality in AMI are age, modified shock index, creatine phosphokinase-MB, peripheral blood leukocyte count, hemoglobin concentration, left ventricular ejection fraction, size of myocardial asynergy, and glomerular filtration rate. The in-hospital mortality model had a high predictive potential: AUC 0.930 (95% CI: 0.905–0.954; $p<0.001$) with a cutoff threshold of 0.15; sensitivity 0.851, and specificity 0.850. |



| | |
|---|---|
| *Keywords* | Myocardial infarction; mortality; prediction |
| *For citations* | Sivkov O.G. Factors Associated With Hospital Mortality in Acute Myocardial Infarction. Kardiologiia. 2023;63(11):29–35. [Russian: Сивков О.Г. Факторы, ассоциированные с госпитальной летальностью при остром инфаркте миокарда. Кардиология. 2023;63(11):29–35]. |


*Corresponding author* Sivkov O. G. E-mail: sivkovog@mail.ru

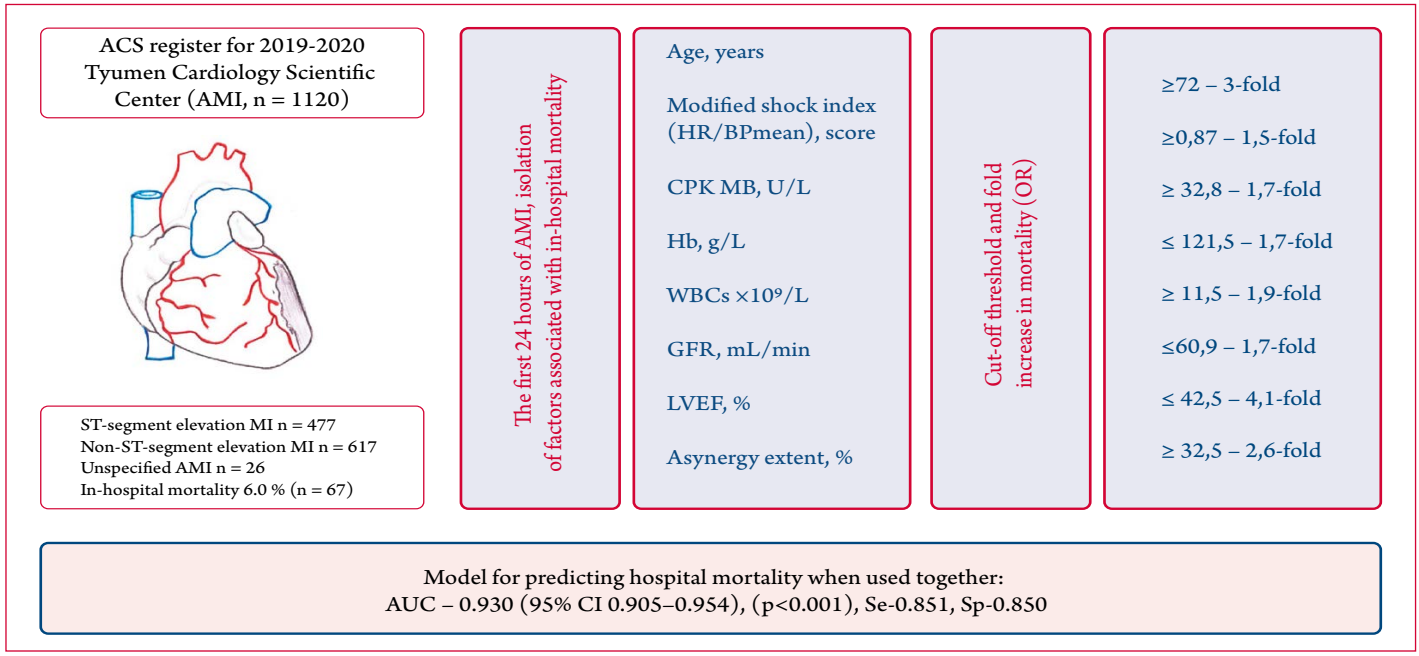
## Introduction

Acute coronary syndrome (ACS) is the most frequent manifestation of cardiovascular pathology with high mortality [1, 2], despite the implementation of modern diagnostic methods, advances in treatment and bringing in measures of primary and secondary prevention. Making critical management decisions regarding the management and treatment of patients with ACS requires risk assessment since patients who are more likely to have adverse clinical events benefit most from aggressive and expensive treatment [3]. Routine risk evaluation of is carried out using various assessment scores. The commonly used scores are the GRACE risk score [3] based on data from the Global Register of Acute Coronary Events, the TIMI score assessing thrombolysis in acute myocardial infarction (AMI) [4], the CADILLAC risk score for identifying patients at low risk of adverse cardiovascular events after ST-segment elevation myocardial infarction who underwent primary percutaneous coronary intervention (PCI) [5], and the PAMI score providing reliable prediction of 30 day in-hospital mortality and 6 month mortality in patients with AMI after primary angioplasty [6]. The best information value of the scores is determined by the creation «geography», since they are adapted to the inclusion and exclusion criteria, patient management strategy, facility logistics and the stream of patients being hospitalized that existed at the time of development. New scoring systems are continually being developed to predict complications in AMI [7]. It is relevant to find the «ideal indicator» that would allow predicting the risk of death for a particular medical facility adapted to its logistics and the existing stream of ACS patients.

## Objective

Determine clinical and laboratory indicators associated with in-hospital mortality in patients with acute myocardial

Central illustration. Factors Associated With Hospital Mortality in Acute Myocardial Infarction




ACS, acute coronary syndrome; AMI, acute myocardial infarction; CI, confidence interval; HR, heart rate, BPmean, mean blood pressure, CPK-MB, creatine phosphokinase MB; WBC, white blood cell; GFR, glomerular filtration rate; LVEF, left ventricular ejection fraction; Hb, hemoglobin; AUC, area under the curve; Se, sensitivity; Sp, specificity.

infarction and develop a multivariate prognostic model of in-hospital mortality.

## Material and methods

Retrospective study of the data of the acute coronary syndrome register of the Tyumen Cardiological Scientific Center, the branch of the Tomsk National Research Medical Center. Signing of the informed consent and using standard approaches to patient management following the existing clinical guidelines were the prerequisites. According to the 2018 ESC Guidelines on Fourth Universal Definition of Myocardial Infarction, AMI was diagnosed based on the results of electrocardiography, serial measurements of cardiac troponin, echocardiography and, if indicated, additional clinical examinations [8]. When objective diagnostic challenges were present, unspecified AMI was established. Of all patients admitted with a referral diagnosis of ACS in 2019 and 2020 (n=1310), a cohort with a confirmed diagnosis of AMI (n=1120) was selected (see Table 1 in supplementary materials in the journal website).

Treatment, including drug and non-drug therapy, nutrition management, anesthesia, medical indications and contraindications to treatments, was conducted in accordance with the current guidelines of the Russian Cardiological Society approved by the Scientific and Practical Council of the Ministry of Health of the Russian Federation. Complete blood count tests were carried out in an XN L 450 automated hematology analyzers (Sysmex, Japan), biochemical tests were performed in a Cobas Intergra 400 plus analyzer (Roche Diagnostics GmbH, Germany), troponin T was determined in a Cobas h 232 express immunochemical analyzer (Germany), and high-sensitivity I was analyzed in the PATHFAST immunochemiluminescent express analyzer (LSI Medience Corporation, Japan). The modified shock index (MSI) was calculated as the ratio of heart rate (HR) to mean blood pressure (BPmean). Two-dimensional (2D) echocardiography was conducted using a General Electric VIvid IQ device (USA). The statistical processing of data was performed in IBM SPSS Statistics v.26. After verification of the distribution normality using the Shapiro-Wilk test, the results were presented as the mean values and standard deviations (M ± σ) or the medians and interquartile ranges (Me (Q25; Q75)). Parametric and non-parametric criteria were used to compare the groups. The cut-off value, sensitivity and specificity of each parameter were determined using ROC analysis. The data set of each quantitative parameter was converted into a binary variable according to the obtained cut-off values, the in-hospital mortality odds ratio was estimated, and a multivariate model for the prognosis of in-hospital mortality was created by stepwise analysis with backward inclusion (Wald's method). The null hypothesis was rejected at $p<0.05$.

## Results

In 2019–2022, 1310 patients with suspected ACS were admitted to the hospital, in-hospital mortality was 8.4% (n=110). Patients with unconfirmed AMI (n=147) and deceased patients (n=43) who did not complete the entire


ISSN 0022-9040. Kardiologiia. 2023;63(11). DOI: 10.18087/cardio.2023.11.n2406

set of routine examinations (mainly patients who died within 24 hours) were excluded from the study. Thus, the study included 1120 patients with AMI, with in-hospital mortality of 6% (n=67) (see Table 1 in the supplementary materials). The immediate cause of death in the study cohort of AMI patients was: acute cardiovascular failure (n=32), progression of multiple organ failure (n=19), cardiogenic shock (n=15), myocardial rupture (n=1). Table 2 (see supplementary materials in the journal website) shows the results of the data comparison between diseased and surviving hospitalized patients. There were no statistically significant differences in the following parameters: body mass index, platelet count, lymphocyte percentage, concentrations of high-density lipoprotein cholesterol and triglycerides.

After determining the cut-off values in the ROC analysis, the data set of each quantitative parameter was converted into a binary variable, and the in-hospital mortality odds ratio was determined using logistic regression (Table 1).

Of the obtained results (Table 1), the highest odds ratio was established for left ventricular ejection fraction (LVEF), because the risk of an adverse outcome increases 15.4-fold in LVEF<42.5%.

A multivariate logistic regression analysis (p<0.001) was carried out to build an optimal model of in-hospital mortality and determine independent predictors of fatal outcomes (Table 2).

The multivariate model evaluated by ROC analysis (Figure 1) had high prognostic potential: AUC 0.930 (95% CI: 0.905–0.954; p<0.001), at a cutoff of 0.15, sensitivity and specificity 0.851 and 0.850, respectively.

**Table 1.** Odds ratio for in-hospital mortality when dividing the indicators according to the cutoff values obtained

| Independent variables | Cutoff value | OR | 95 % confidence interval | | p |
|---|---|---|---|---|---|
| | | | Lower limit | Upper limit | |
| Age, years | ≥71.5 | 2.109 | 1.116 | 3.987 | 0.001 |
| MSI | ≥0.87 | 2.161 | 1.568 | 4.367 | <0.001 |
| CPK, U/L | ≥234.7 | 3.398 | 2.054 | 5.621 | <0.001 |
| CPK MB, U/L | ≥32.8 | 8.928 | 5.281 | 15.092 | <0.001 |
| RBCs, $\times 10^{12}$/L | ≤4.27 | 1.901 | 1.176 | 3.074 | 0.009 |
| Hemoglobin, g/L | ≤121.5 | 2.564 | 1.592 | 4.423 | <0.001 |
| WBCs, $10^9$/L | ≥11.5 | 4.306 | 2.58 | 7.186 | <0.001 |
| Cholesterol, mmol/L | ≥5.2 | 2.178 | 1.252 | 3.789 | 0.006 |
| LDL cholesterol, mmol/L | ≥3.1 | 0.037 | 0.343 | 0.968 | 0.037 |
| GFR MDRD, mL/min | ≤60.9 | 3.435 | 2.07 | 5.701 | <0.001 |
| Left ventricular ejection fraction, % | ≤42.5 | 15.385 | 8.663 | 27.32 | <0.001 |
| Asynergy extent, % | ≥32.5 | 7.003 | 4.111 | 11.93 | <0.001 |
| Glycemia, mmol/L | ≥9.1 | 4.413 | 2.668 | 7.3 | <0.001 |

OR, odds ratio; MSI, modified shock index; CPK MB, creatine phosphokinase MB; GFR, glomerular filtration rate; LDL, low-density lipoproteins.

## Discussion

It was established that age and sex are closely associated with mortality in myocardial infarction mainly due to the «contribution» of older female patients [9]. According to the results obtained, the risk of in-hospital mortality is 2.4 times higher in female patients than in male patients. The age of female subjects 70 (63; 82) years was statistically significantly higher (p<0.001) than the age of male subjects 61 (55; 96) years. The age of≥72 years was associated in AMI, irrespective of sex, with a 2.1-fold increase in in-hospital mortality.

MSI is an easily accessible indicator that does not depend on subjective data, ECG findings or blood tests [10], it is also a clinically significant predictor of in-hospital mortality in emergency patients. Each of its components is closely associated with the risk of death in patients with ACS. As well as systolic blood pressure [11], lower SBP at admission (SBP<79 mm Hg) was associated with a higher rate of nosocomial mortality in post-primary-PCI patients [12]. Observational study by Dobre et al., which included 22,398

**Table 2.** Model for the prognosis of in-hospital mortality in patients with AMI

| Independent variables | B | OR | 95 % confidence interval | | p |
|---|---|---|---|---|---|
| | | | Lower limit | Upper limit | |
| Age, years | 1,096 | 2,991 | 1,606 | 5,569 | 0,001 |
| MSI | 0,374 | 1,453 | 1,067 | 1,980 | 0,018 |
| CPK MB, U/L | 1,417 | 4,124 | 2,195 | 7,747 | <0,001 |
| Hemoglobin, g/L | 0,502 | 1,652 | 1,165 | 2,343 | 0,005 |
| WBCs, 109/L | 0,616 | 1,851 | 1,296 | 2,644 | 0,001 |
| GFR MDRD, mL/min | 0,501 | 1,650 | 1,223 | 2,226 | 0,001 |
| Left ventricular ejection fraction, % | 1,400 | 4,055 | 1,992 | 8,255 | <0,001 |
| Asynergy extent, % | 0,967 | 2,630 | 1,397 | 4,949 | 0,003 |
| Constant | –12,578 | – | – | – | <0,001 |

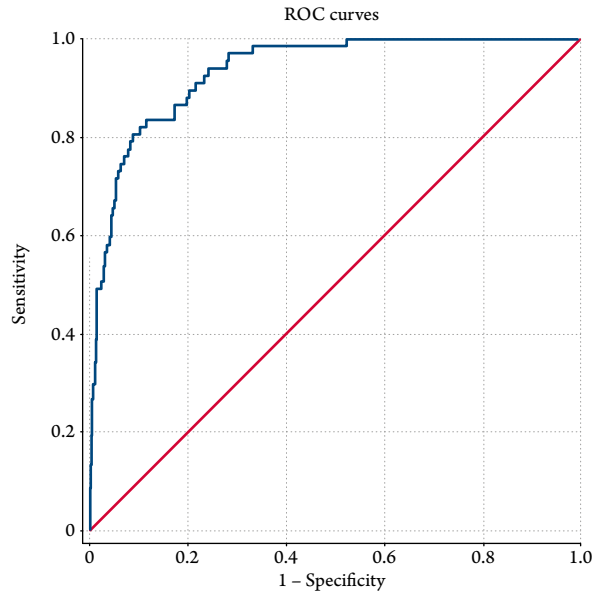
MSI, modified shock index; CPK MB, creatine phosphokinase MB; GFR, glomerular filtration rate.

patients with AMI and heart failure, showed that a higher HR was independently associated with all-cause mortality and cardiovascular mortality [13]. The effectiveness of MSI for the prognosis of in-hospital mortality was proven in STEMI patients [14] at a cutoff of 0.91, sensitivity and specificity 80.0 and 56.2, respectively. The cutoff threshold was 0.86 with corresponding sensitivity and specificity of 0.716 and 0.797, respectively. Values higher than the cutoff threshold increased 2.2-fold the risk of in-hospital mortality. Several explanations were proposed for the association between MSI and mortality in ACS patients [15]. Firstly, SBP may be indicative of any deterioration in left ventricular function – a decrease in stroke volume and cardiac index [16] as a result of extensive left ventricular remodeling and heart failure, which is accompanied by higher mortality [15]. Secondly, a higher HR may be indicative of sympathetic nervous hyperactivity, which leads to more severe LV dysfunction [17] and fatal ventricular arrhythmias, which are common complications with a significant percentage of sudden cardiac death [18]. However, despite the fact that MSI is practical and convenient indicator, it has a lower prognostic value compared to the GRACE score [19]. Moreover, the association between HR and in-hospital mortality is J-shaped rather than linear [20]. Bangalore et al. [20] showed that mortality increases at HR<50 bpm and > 130 bpm.

The concentration of creatine phosphokinase (CPK) MB increases rapidly in ACS in the first 4–8 hours, reaches a peak in 12–24 hours, and decreases to baseline within 3–4 days. The existing studies assess the possibility of predicting in-hospital 3 [21] and 6-month [22] mortality by peak values of the indicator. Unlike in earlier works, the levels of CPK and CPK MB were assessed in our study within the first 3 hours after hospitalization, and both indicators were associated with in-hospital mortality. Elevated CPK > 234.7 U/L and CPK MB > 32.8 were associated with the 3.4-fold and 8.9-fold risk of an adverse outcome, respectively.

Blood transfusion is associated with an increase in mortality in ACS [23]. However, McKechnie et al. (2004) made an extremely important conclusion that anemia, rather than blood transfusion, is an independent risk factor for adverse cardiovascular outcomes in patients with heart failure and in patients with history of PCI [24]. They showed that patients with anemia have a more pronounced comorbid background, which results in more severe course of AMI with increased in-hospital mortality in this group. More recent studies have confirmed that female sex, antiplatelet drug therapy, and elevated creatinine are associated with anemia, which is in turn associated with adverse hospital outcomes [25]. In another study, anemic patients with history of PCI associated with clinical stent restenosis and an increased incidence of major adverse

**Figure 1.** ROC curve of the multivariate model for the prognosis of in-hospital mortality in patients with AMI




cardiac events (MACE) were mainly female patients with diabetes mellitus and renal failure [26]. The risk of MACE in AMI increased 1.45 times (95% CI: 1.33–1.58) per 1 g/dL decrease in hemoglobin below 11 g/dL [27]. STEMI patients with history of PCI and low hemoglobin before surgery were more likely to develop MACE, especially if they had history of AMI [28]. There are no validated guidelines for initiating blood transfusion in AMI. The dilemma between restrictive and liberal blood transfusion strategies in AMI patients is determined by the lack of well-defined hemoglobin level boundaries for these strategies. In existing studies, the liberal and restrictive strategies are defined by different hemoglobin levels from 70 g/L to 140 g/L [29]. In our study, a decrease in red blood cell count of less than 4.2×1012/L and hemoglobin less than 121.5 g/L increased 1.9-fold and 2.6-fold, respectively, the odds of an unfavorable in-hospital outcome in patients with AMI.

Immediately after the onset of ischemia, endothelial cells activate adhesion molecules, which, together with chemokines produced, cause extravasation of neutrophils and their early peak blood level is recorded as soon as 15 minutes after myocardial ischemia [30], with a subsequent maximum in 12 hours [31]. WBC count and the course of ACS are closely related [32]. Barron et al (2000) were the first to document the relation between all-cause death and WBC count in acute MI survivors [33]. In a subsequent large study Cooperative Cardiovascular Project [34] including 150,000 patients above 65 years old who had AMI (60% with non-ST-segment elevation MI or left bundle branch block), the authors showed that patients with elevated white blood cell counts were significantly more likely to die on day 30 than patients with low white blood cell counts. Furman et al. [35] confirmed the


ISSN 0022-9040. Kardiologiia. 2023;63(11). DOI: 10.18087/cardio.2023.11.n2406

relationship between white blood cell count and outcomes in ACS. They found that increased white blood cell counts were associated with in-hospital mortality and the development of heart failure. On this ground, they concluded that the baseline white blood cell count is an independent predictor of in-hospital mortality and the development of heart failure in male and female patients of all ages with diseases of the entire ACS spectrum. According to their findings, the risk of in-hospital mortality increased 4.3-fold in patients with white blood cell count of more than 11.5 thousand.

Reduced LVEF is a serious complication of AMI. There is a correlation between this complication and increased mortality in AMI patients [36]. LVEF is an independent predictor of nosocomial and annual mortality in patients with ST-segment elevation AMI, even when adjusted for the TIMI risk score and other risk factors [36]. CADILLAC was one of the first scores that included LVEF as one of the factors [5], the cutoff was LVEF<40%. The new score developed by Kim et al., which includes LVEF and other predictors of in-hospital mortality, has a greater predictive power than the GRACE score [37]. LVEF<42.5% was associated with a 15.3-fold increase in in-hospital mortality.

GFR is a known risk factor of cardiovascular death [38]. Dohi et al. showed that kidney disease is a factor of long-term all-cause and cardiovascular mortality after ACS [39]. Several other studies also showed that chronic renal failure is an independent predictor of adverse outcomes after PCI [40] associated with an increased risk of nosocomial mortality in ACS [41]. GFR ≤ 60.9 mL/min was associated in our study with a 3.4-fold increase in mortality.

The main advantages of 2D echocardiography are its availability, mobility, relative low cost, non-invasive nature, and ease of use. 2D echocardiography makes it possible to assess local contractility abnormalities. The method developed by Widimsky et al. [42] was used in the study to determine the extent of asynergy. They proposed in 1985 to conditionally divide the left ventricular (LV) myocardium into 10 segments. Each segment corresponds to 10% of the LV mass – 4 basal segments, 4 mid segments, and 2 apical segments. The method allows assessing the severity of local contractility abnormalities without differential diagnosis between cicatricial changes and new necrotic foci, which is more important for predicting unfavorable outcomes.

## Conclusions

Independent predictors of in-hospital mortality in AMI are age, MSI, CPK MB, peripheral blood white blood cell count, hemoglobin concentration, left ventricular ejection fraction, myocardial asynergy extent, and glomerular filtration rate. The model of their joint use has high prognostic potential AUC 0.930 (95% CI: 0.905–0.954; p<0.001), at a cutoff of 0.15, sensitivity and specificity 0.851 and 0.850, respectively.

### *Limitations*

The strengths of the study include the fact that the results obtained are adapted for a particular facility considering its logistics and patient stream. The limitations include a single-center design, the absence of the analysis of the association of troponin concentration and mortality due to the fact that both troponin T and troponin I were determined in this period; the absence of the comparison with the TIMI and GRACE scores due to the fact that they were not used in every study. Patients who died within the first 24 hours were excluded from the study. On the one hand, this improves the accuracy of the model for patients who survived the first day, and on the other hand, it reduces the accuracy of prognosis for assessing overall mortality in AMI.

*No conflict of interest is reported.*

**The article was received on 18/01/2023**

REFERENCES


1. Roffi M, Patrono C, Collet J-P, Mueller C, Valgimigli M, Andreotti F et al. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). European Heart Journal. 2016;37(3):267–315. DOI: 10.1093/eurheartj/ehv320
2. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). European Heart Journal. 2018;39(2):119–77. DOI: 10.1093/eurheartj/ehx393
3. Granger CB. Predictors of Hospital Mortality in the Global Registry of Acute Coronary Events. Archives of Internal Medicine. 2003;163(19):2345–53. DOI: 10.1001/archinte.163.19.2345
4. Antman EM, Cohen M, Bernink PJLM, McCabe CH, Horacek T, Papuchis G et al. The TIMI Risk Score for Unstable Angina/Non–ST Elevation MI: A Method for Prognostication and Therapeutic Decision Making. JAMA. 2000;284(7):835–42. DOI: 10.1001/jama.284.7.835
5. Halkin A, Singh M, Nikolsky E, Grines CL, Tcheng JE, Garcia E et al. Prediction of Mortality After Primary Percutaneous Coronary Intervention for Acute Myocardial Infarction: The CADILLAC risk score. Journal of the American College of Cardiology. 2005;45(9):1397–405. DOI: 10.1016/j.jacc.2005.01.041
6. Baptista SB, Farto e Abreu P, Loureiro JR, Thomas B, Nédio M, Gago S et al. PAMI risk score for mortality prediction in acute myocardial indarction treated with primary angioplasty. Portuguese Journal of Cardiology. 2004;23(5):683–93. PMID: 15279453
7. Bessonov I.S., Kuznetsov V.A., Sapozhnikov S.S., Gorbatenko E.A., Shadrin A.A. The risk score for in-hospital mortality in patients with ST-segment elevation myocardial infarction. Kardiologiia. 2021;61(9):11–9. [Russian: Бессонов И.С.,

Кузнецов В.А., Сапожников С.С., Горбатенко Е.А., Шадрин А.А. Шкала оценки риска госпитальной летальности у пациентов с острым инфарктом миокарда с подъемом сегмента ST электрокардиограммы. Кардиология. 2021;61(9):11-9]. DOI: 10.18087/cardio.2021.9.n1720
8. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA et al. Fourth Universal Definition of Myocardial Infarction (2018). Journal of the American College of Cardiology. 2018;72(18):2231–64. DOI: 10.1016/j.jacc.2018.08.1038
9. Gupta T, Kolte D, Khera S, Agarwal N, Villablanca PA, Goel K et al. Contemporary Sex-Based Differences by Age in Presenting Characteristics, Use of an Early Invasive Strategy, and Inhospital Mortality in Patients With Non–ST-Segment–Elevation Myocardial Infarction in the United States. Circulation: Cardiovascular Interventions. 2018;11(1):e005735. DOI: 10.1161/CIRCINTERVENTIONS.117.005735
10. Liu Y-C, Liu J-H, Fang ZA, Shan G-L, Xu J, Qi Z-W et al. Modified shock index and mortality rate of emergency patients. World Journal of Emergency Medicine. 2012;3(2):114–7. DOI: 10.5847/wjem.j.issn.1920-8642.2012.02.006
11. Pei J, Wang X, Xing Z, Chen P, Su W, Deng S et al. Association between admission systolic blood pressure and major adverse cardiovascular events in patients with acute myocardial infarction. PLOS ONE. 2020;15(6):e0234935. DOI: 10.1371/journal.pone.0234935
12. Shiraishi J, Nakamura T, Shikuma A, Shoji K, Nishikawa M, Yanagiuchi T et al. Relationship Between Mean Blood Pressure at Admission and In-Hospital Outcome After Primary Percutaneous Coronary Intervention for Acute Myocardial Infarction. International Heart Journal. 2016;57(5):547–52. DOI: 10.1536/ihj.15-480
13. Dobre D, Kjekshus J, Rossignol P, Girerd N, Benetos A, Dickstein K et al. Heart rate, pulse pressure and mortality in patients with myocardial infarction complicated by heart failure. International Journal of Cardiology. 2018;271:181–5. DOI: 10.1016/j.ijcard.2018.05.017
14. Gouda M, Saad AM, Al-Daydamony MM. Modified Shock Index as a Predictor of In-Hospital Outcome in Cases of ST-Segment Elevation Myocardial Infarction Treated with Primary Percutaneous Coronary Intervention. Journal of Cardiology & Current Research. 2016;7(4):11–2. DOI: 10.15406/jccr.2016.07.00255
15. Yu T, Tian C, Song J, He D, Sun Z, Sun Z. Derivation and Validation of Shock Index as a parameter for Predicting Long-term Prognosis in Patients with Acute Coronary Syndrome. Scientific Reports. 2017;7(1):11929. DOI: 10.1038/s41598-017-12180-2
16. Rady MY, Nightingale P, Little RA, Edwards JD. Shock index: a re-evaluation in acute circulatory failure. Resuscitation. 1992;23(3):227–34. DOI: 10.1016/0300-9572(92)90006-X
17. Graham LN, Smith PA, Stoker JB, Mackintosh AF, Mary DA. Sympathetic neural hyperactivity and its normalization following unstable angina and acute myocardial infarction. Clinical Science. 2004;106(6):605–11. DOI: 10.1042/CS20030376
18. Chen P, Chen LS, Cao JM, Sharifi B, Karagueuzian HS, Fishbein MC. Sympathetic nerve sprouting, electrical remodeling and the mechanisms of sudden cardiac death. Cardiovascular Research. 2001;50(2):409–16. DOI: 10.1016/S0008-6363(00)00308-4
19. Pramudyo M, Marindani V, Achmad C, Putra ICS. Modified Shock Index as Simple Clinical Independent Predictor of In-Hospital Mortality in Acute Coronary Syndrome Patients: A Retrospective Cohort Study. Frontiers in Cardiovascular Medicine. 2022;9:915881. DOI: 10.3389/fcvm.2022.915881
20. Bangalore S, Messerli FH, Ou F-S, Tamis-Holland J, Palazzo A, Roe MT et al. The association of admission heart rate and in-hospital cardiovascular events in patients with non-ST-segment elevation acute coronary syndromes: results from 135 164 patients in the CRUSADE quality improvement initiative. European Heart Journal. 2010;31(5):552–60. DOI: 10.1093/eurheartj/ehp397
21. Chin CT, Wang TY, Li S, Wiviott SD, deLemos JA, Kontos MC et al. Comparison of the Prognostic Value of Peak Creatine Kinase-MB and Troponin Levels Among Patients With Acute Myocardial Infarction: A Report from the Acute Coronary Treatment and Intervention Outcomes Network Registry-Get With The Guidelines. Clinical Cardiology. 2012;35(7):424–9. DOI: 10.1002/clc.21980
22. Bagai A, Schulte PJ, Granger CB, Mahaffey KW, Christenson RH, Bell G et al. Prognostic implications of creatine kinase–MB measurements in ST-segment elevation myocardial infarction patients treated with primary percutaneous coronary intervention. American Heart Journal. 2014;168(4):503-511.e2. DOI: 10.1016/j.ahj.2014.06.008
23. Yin Z, Yu B, Liu W, Lan K. Blood transfusion and mortality in myocardial infarction: an updated meta-analysis. Oncotarget. 2017;8(60):102254–62. DOI: 10.18632/oncotarget.19208
24. McKechnie RS, Smith D, Montoye C, Kline-Rogers E, O'Donnell MJ, DeFranco AC et al. Prognostic implication of anemia on in-hospital outcomes after percutaneous coronary intervention. Circulation. 2004;110(3):271–7. DOI: 10.1161/01.CIR.0000134964.01697.C7
25. Bhavanadhar P, Srinivasan VR, Oruganti SS, Adiraju KP. A Prospective Study on Prevalence and Causes of Anaemia in Patients with Acute Coronary Syndrome. Journal of clinical and diagnostic research. 2016;10(7):OC01-5. DOI: 10.7860/JCDR/2016/19091.8106
26. Hussein A, Awad MS, Sabra AM, Mahmoud HEM. Anemia is a novel predictor for clinical ISR following PCI. The Egyptian Heart Journal. 2021;73(1):40. DOI: 10.1186/s43044-021-00163-8
27. Sabatine MS, Morrow DA, Giugliano RP, Burton PBJ, Murphy SA, McCabe CH et al. Association of Hemoglobin Levels With Clinical Outcomes in Acute Coronary Syndromes. Circulation. 2005;111(16):2042–9. DOI: 10.1161/01.CIR.0000162477.70955.5F
28. Yang Y, Huang Y. Association between serum hemoglobin and major cardiovascular adverse event in Chinese patients with ST-segment elevation myocardial infarction after percutaneous coronary intervention. Journal of Clinical Laboratory Analysis. 2022;36(1):e24126. DOI: 10.1002/jcla.24126
29. Ducrocq G, Gonzalez-Juanatey JR, Puymirat E, Lemesle G, Cachanado M, Durand-Zaleski I et al. Effect of a Restrictive vs Liberal Blood Transfusion Strategy on Major Cardiovascular Events Among Patients With Acute Myocardial Infarction and Anemia: The REALITY Randomized Clinical Trial. JAMA. 2021;325(6):552–60. DOI: 10.1001/jama.2021.0135
30. Liebetrau C, Hoffmann J, Dörr O, Gaede L, Blumenstein J, Biermann H et al. Release Kinetics of Inflammatory Biomarkers in a Clinical Model of Acute Myocardial Infarction. Circulation Research. 2015;116(5):867–75. DOI: 10.1161/CIRCRESAHA.116.304653
31. Husser O, Bodi V, Sanchis J, Nunez J, Mainar L, Chorro FJ et al. White Blood Cell Subtypes after STEMI: Temporal Evolution, Association with Cardiovascular Magnetic Resonance – Derived Infarct Size and Impact on Outcome. Inflammation. 2011;34(2):73–84. DOI: 10.1007/s10753-010-9209-0
32. Bodí V, Llacer A, Sanchis J, Nunez J, Nunez E. Prognostic Value of Leukocytosis in Acute Coronary Syndromes: The Cinderella of the Inflammatory Markers. Current Medicinal Chemistry. 2006;13(18):2113–8. DOI: 10.2174/092986706777935221
33. Barron HV, Cannon CP, Murphy SA, Braunwald E, Gibson CM. Association Between White Blood Cell Count, Epicardial Blood Flow, Myocardial Perfusion, and Clinical Outcomes in the Setting of Acute Myocardial Infarction: A Thrombolysis In Myocardial Infarction 10 Substudy. Circulation. 2000;102(19):2329–34. DOI: 10.1161/01.CIR.102.19.2329
34. Barron HV, Harr SD, Radford MJ, Wang Y, Krumholz HM. The association between white blood cell count and acute myocardial infarction mortality in patients ≥65 years of age: findings from the cooperative cardiovascular project. Journal of the American College of Cardiology. 2001;38(6):1654–61. DOI: 10.1016/S0735-1097(01)01613-8
35. Furman MI, Gore JM, Anderson FA, Budaj A, Goodman SG, Avezum Á et al. Elevated leukocyte count and adverse hospital events in patients with acute coronary syndromes: findings from the Global Registry of Acute Coronary Events (GRACE). American Heart Journal. 2004;147(1):42–8. DOI: 10.1016/j.ahj.2003.07.003
36. Wei X-B, Liu Y-H, He P-C, Jiang L, Zhou Y-L, Chen J-Y et al. Additive prognostic value of left ventricular ejection fraction to the TIMI


ISSN 0022-9040. Kardiologiia. 2023;63(11). DOI: 10.18087/cardio.2023.11.n2406

risk score for in-hospital and long-term mortality in patients with ST segment elevation myocardial infarction. Journal of Thrombosis and Thrombolysis. 2017;43(1):1–6. DOI: 10.1007/s11239-016-1407-7
37. Kim HK, Jeong MH, Ahn Y, Kim JH, Chae SC, Kim YJ et al. Hospital discharge risk score system for the assessment of clinical outcomes in patients with acute myocardial infarction (Korea Acute Myocardial Infarction Registry [KAMIR] score). The American Journal of Cardiology. 2011;107(7):965-971.e1. DOI: 10.1016/j.amjcard.2010.11.018
38. Khwaja A. KDIGO Clinical Practice Guidelines for Acute Kidney Injury. Nephron Clinical Practice. 2012;120(4):c179–84. DOI: 10.1159/000339789
39. Dohi T, Kasai T, Miyauchi K, Takasu K, Kajimoto K, Kubota N et al. Prognostic impact of chronic kidney disease on 10-year clinical outcomes among patients with acute coronary syndrome. Journal of Cardiology. 2012;60(6):438–42. DOI: 10.1016/j.jjcc.2012.08.007
40. Gruberg L, Dangas G, Mehran R, Mintz GS, Kent KM, Pichard AD et al. Clinical outcome following percutaneous coronary interventions in patients with chronic renal failure. Catheterization and Cardiovascular Interventions. 2002;55(1):66–72. DOI: 10.1002/ccd.10103
41. Lenci Marques G, Assano Stangler NH, Ferro H, Calisto J, Brehm J, Felicio Morais G et al. Kidney Disease as Risk of In-Hospital Mortality in Patients With Acute Coronary Syndrome. Cureus. 2021;13(11):e19557. DOI: 10.7759/cureus.19557
42. Widimský P, Gregor P, Cervenka V, Vísek V. The role of echocardiography in a coronary care unit. Cor Et Vasa. 1985;27(4):272–9. PMID: 4053618

Sivkov O. G.
Surgut State University, Surgut, Khanty-Mansi Autonomous District-Ugra, Russia

# Factors Associated With Hospital Mortality in Acute Myocardial Infarction

# ADDITIONAL MATERIALS

**Table 1.** Characteristics of patients with myocardial infarction (n = 1120) in 2019–2020

| Parameter | 2019 (n=457) | 2020 (n=663) | 2019 и 2020 (n=1120) |
|---|---|---|---|
| Days in the hospital (all patients) | 10 (8;13) | 9 (7;12) | 10 (8;12) |
| Days in the hospital (surviving patients) | 10 (8;13) | 10 (8;12) | 10 (8;12) |
| Days in the hospital (deceased patients) | 3 (0;7) | 2.5 (0;6) | 3 (0;6) |
| History of myocardial infarction, n (%) | 153 (33.3) | 202(30.4) | 35(31.7) |
| History of CAD, n (%) | 216 (47.3) | 381(57.5) | 597 (53) |
| AF, n (%) | 59 (12.9) | 99 (14.9) | 158 (14.1) |
| VF, n (%) | 12 (2.5) | 3 (0.5) | 15 (1.2) |
| Structural heart defects, n (%) | 32 (6.9) | 30 (4.5) | 62 (5.1) |
| History of CVA, n (%) | 40 (8.7) | 15 (2.2) | 55 (4.9) |
| History of HHD, n (%) | 402 (87.9) | 567 (85.5) | 969 (86.5) |
| CHF, n (%) | 452 (98.9) | 661(99.7) | 1113 (99.6) |
| History of DM, n (%) | 120 (26.1) | 128 (20.8) | 258 (23.3) |
| ST-elevation myocardial infarction, n (%) | 167 (36.7) | 310 (46.8) | 477 (42.7) |
| Non-ST-elevation myocardial infarction, n (%) | 266 (58.0) | 352 (53.2) | 618 (55.2) |
| Myocardial infarction, unspecified, n (%) | 24 (5.3) | – | 24 (2.1) |
| Coronary angiography | | | |
| All patients, n (%) | 412 (90.1) | 601 (90.6) | 1013 (90.4) |
| ST-elevation myocardial infarction, n (%) | 421 (92.2) | 614 (92.6) | 1035 (92.4) |
| Non-ST-elevation myocardial infarction, n (%) | 410 (89.8) | 591 (89.2) | 1001(89.6) |
| Myocardial infarction, unspecified, n (%) | 280 (61.3) | – | 280 (25) |
| Percutaneous coronary intervention | | | |
| All patients, n (%) | 147 (37.2) | 374 (56.4) | 521 (51.6) |
| ST-elevation myocardial infarction, n (%) | 326 (71.3) | 534 (80.6) | 860 (77.4) |
| Non-ST-elevation myocardial infarction, n (%) | 81 (17.7) | 233 (35.1) | 314 (27.7) |
| Myocardial infarction, unspecified, n (%) | 59 (12.9) | – | 59 (5.0) |
| Mortality, AMI (n = 1120), n (%) | 17 (3.6) | 50 (7.6) | 67 (6.0) |
| Mortality, ACS (n = 1310), n (%) | 50 (10.1) | 60 (7.4) | 110 (8.4) |

The data are expressed as the medians and interquartile ranges (Me [Q25; Q75]), and the absolute and relative numbers of patients (n (%)).
CAD, coronary artery disease; AF, atrial fibrillation; VF, ventricular fibrillation; CVA, cerebrovascular accident; HHD, hypertensive heart disease; CHF, chronic heart failure; DM, diabetes mellitus; ACS, acute coronary syndrome; AMI, acute myocardial infarction.

**Table 2.** Clinical and demographic characteristics and results of laboratory tests and clinical examinations of patients with AMI

| Parameter | All patients, n = 1120 | Deceased patients, n = 67 | Surviving patients, n = 1053 | p (deceased and surviving patient groups) |
|---|---|---|---|---|
| Sex, M/F | 61.8/38.2 | 41.8/58.2 | 63.1/36.9 | 0.001 |
| Age, years | 64 (57; 72) | 72 (64; 84) | 63 (57.0; 71.0) | <0.001 |
| BMI, kg/m$^2$ | 28.7 (25.4; 32) | 27.3 (24.1; 34.6) | 28.7 (25.6; 31.9) | 0.516 |
| MSI | 0.72 (0.63; 0.85) | 1.1 (0.8; 1.5) | 0.7 (0.6; 0.8) | <0.001 |
| CPK, U/L | 125 (83; 229) | 315 (150; 1048) | 122 (82; 213) | <0.001 |
| CPK MB, U/L | 15.9 (11.8; 25.8) | 42.6 (19.4; 98.0) | 15.6 (11.7; 23.7) | <0.001 |
| Erythrocytes, ×1012/L | 4.7 (4.3; 5.1) | 4.3 (3.8; 4.9) | 4.7 (4.3; 5.1) | <0.001 |
| Hemoglobin, g/L | 136 (123; 147) | 120.7±23.7 | 137 (124; 148) | <0.001 |
| Leukocytes, ×10$^9$/L | 8.2 (6.4; 10.4) | 12.1 (9.1; 15.5) | 8.0 (6.4; 10.1) | <0.001 |
| Platelets, ×10$^9$/L | 219 (186; 261) | 220 (172; 257) | 219 (186; 261) | 0.421 |
| Lymphocytes, % | 1.8 (1.3; 2.4) | 1.7 (1.1; 3.0) | 1.8 (1.4; 2.3) | 0.47 |
| Cholesterol, mmol/L | 5 (4.0; 5.9) | 4.6±1.3 | 5.0 (4.1; 5.9) | 0.011 |
| HDL cholesterol, mmol/L | 1.2 (1.0; 1.4) | 1.1 (0.9; 1.4) | 1.2 (1.0; 1.4) | 0.137 |
| LDL cholesterol, mmol/L | 3.1 (2.3; 3.8) | 2.8±1.0 | 3.1 (2.4; 3.9) | 0.033 |
| Triglycerides, mmol/L | 1.27 (0.93; 1.8) | 1.28 (1.0; 1.8) | 1.27 (0.9; 1.8) | 0.656 |
| GFR MDRD, mL/min | 75.3 (58.8; 91.3) | 47.4 (32.0; 58.4) | 76.9 (62.2; 92.4) | <0.001 |
| Left ventricular ejection fraction, % | 54 (45; 59) | 38 (33.5; 43.0) | 54 (46.0; 60.0) | <0.001 |
| Asynergy extent, % | 20 (0; 35) | 40 (35.0; 42.0) | 20 (0; 30.0) | <0.001 |
| Glycemia, mmol/L | 6.8 (5.9; 9.0) | 9.5 (6.2; 12.1) | 6.7 (5.9; 8.6) | <0.001 |

The data are expressed as the medians and interquartile ranges (Me [Q25; Q75]). BMI, body mass index;
MSI, modified shock index; CPK, creatine phosphokinase; CPK MB, creatine phosphokinase MB fraction; GFR, glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein


ISSN 0022-9040. Kardiologiia. 2023;63(11). DOI: 10.18087/cardio.2023.11.n2406