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ФАКТОРЫ РИСКА ГОСПИТАЛЬНОЙ ЛЕТАЛЬНОСТИ ПРИ ОСТРОМ КОРОНАРНОМ СИНДРОМЕ С ПОДЪЕМОМ СЕГМЕНТА ST, ОСЛОЖНЕННОМ КАРДИОГЕННЫМ ШОКОМ

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Risk Factors for Hospital Mortality in Acute ST-Segment Elevation Coronary Syndrome Complicated by Cardiogenic Shock

Резюме

Цель исследования. Изучить факторы риска госпитальной летальности у больных острым коронарным синдромом с подъемом сегмента ST (ОКСпST), осложненным кардиогенным шоком (КШ). **Материал и методы.** Исследовались 104 пациента с ОКСпST, осложненным КШ. В группу наблюдения (I группу) вошли 58 (55,8%) умерших в стационаре больных (средний возраст $71,8 \pm 7,31$ лет), в группу сравнения (II группу) — 46 (44,2%) пациентов, прошедших лечение и выписавшихся (средний возраст $59,5 \pm 6,18$ лет). Всем больным проводились общеклинические исследования, определялся уровень тропонинов, липидов, глюкозы, креатинина плазмы, выполнялась электрокардиография и эхокардиография. Экстренно проводили коронароангиографию и чрескожное коронарное вмешательство (ЧКВ). Для выявления факторов риска госпитальной летальности использовали метод бинарной логистической регрессии с определением для каждой достоверной переменной отношения шансов и его 95% доверительного интервала. **Результаты.** В I группе больных с КШ, по сравнению со II группой, значимо чаще наблюдались пациенты в возрасте старше 70 лет (32 (55,2%) vs 10 (22,7%), $p=0,0004$), с сопутствующей хронической болезнью почек (32 (55,2%) vs 9 (19,6%), $p=0,0002$), постинфарктным кардиосклерозом (30 (51,7%) vs 9 (19,6%), $p=0,001$) и хронической сердечной недостаточностью III-IV функционального класса (32 (55,1%) vs 11 (23,9%), $p=0,001$). Исходные уровни лейкоцитов, тропонина и креатинина плазмы были достоверно выше у умерших больных с КШ. Фракция выброса левого желудочка ниже 40% отмечалась чаще в группе наблюдения, чем в группе сравнения (46 (79,3%) vs 27 (58,7%), $p=0,022$). В I группе, по сравнению со II группой, была выше частота трехсосудистого поражения венечного русла (36 (75%) vs 12 (26,1%), $p=0,0001$) и хронической окклюзии коронарной артерии, несвязанной с ОКСпST (25 (52,1%) vs 12 (26,1%), $p=0,009$). Такая же тенденция отмечалась при оценке среднего числа стенозов и окклюзий коронарных артерий. ЧКВ выполнено 43 (74,1%) умершим и 43 (93,5%) выжившим больным ОКСпST с КШ ($p=0,009$). В группе наблюдения, чем в группе сравнения, была выше частота безуспешного ЧКВ (13 (30,2%) vs 3 (7%), $p=0,001$) и проведенного позднее 6 часов от начала ангинозного приступа (28 (65,1%) vs 6 (14%), $p=0,0001$). **Выводы.** Госпитальная летальность у больных ОКСпST, осложненным КШ, ассоциировалась с наличием у них фракции выброса левого желудочка менее 40%, трехсосудистого поражения коронарного русла и проведением ЧКВ позднее 6 часов от начала болевого приступа.

Ключевые слова: кардиогенный шок, острый коронарный синдром с подъемом сегмента ST, госпитальная летальность, факторы риска, предикторы, прогноз

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

The aim. To study the risk factors for hospital mortality in patients with acute coronary syndrome with ST-segment elevation (STEACS) complicated by cardiogenic shock (CS). **Materials and methods.** A total of 104 patients with STEACS complicated by CS were studied. The follow-up group (group I) included 58 (55,8%) patients who died in hospital (mean age $71,8 \pm 7,31$ years), the comparison group (group II) — 46 patients, who have been treated and discharged (mean age $59,5 \pm 6,18$ years). All patients underwent general clinical studies, the level of troponins, lipids, glucose, creatinine in plasma was determined, electrocardiography and echocardiography were performed. Coronary angiography and percutaneous coronary intervention (PCI) were urgently performed. The method of binary logistic regression with the determination of the odds ratio and its 95% confidence interval for each reliable variable was used to identify risk factors for hospital mortality. **Results.** In group I patients with CS, compared with group II, patients over the age of 70 (32 (55,2%) vs 10 (22,7%), $p=0,0004$), with concomitant chronic kidney disease (32 (55,2%) vs 9 (19,6%), $p=0,0002$), postinfarction cardiosclerosis (30 (51,7%) vs 9 (19,6%), $p=0,001$) and chronic heart failure of III-IV functional class (32 (55,1%) vs 11 (23,9%), $p=0,001$) were significantly more often observed. Baseline levels of plasma leukocytes, troponin and creatinine were significantly higher in deceased patients with CS. Left ventricular ejection fraction below 40% was observed more often in the follow-up group than in the comparison group (46 (79,3%) vs 27 (58,7%), $p=0,022$). In group I, compared with group II, there was a higher incidence of three-vessel coronary lesions (36 (75%) vs 12 (26,1%), $p=0,0001$) and chronic coronary artery occlusion unrelated to STEACS (25 (52,1%) vs 12 (26,1%), $p=0,009$). The same trend was observed when assessing the average number of stenoses and occlusions of the coronary arteries. PCI was performed in 43 (74,1%) of the deceased and 43 (93,5%) of the surviving STEACS patients with CS ($p=0,009$). The follow-up group had a higher rate of unsuccessful PCI (30,2%) vs 3 (7%), $p=0,001$ and performed later than 6 hours after the onset of an angina attack (28 (65,1%) vs 6 (14%), $p=0,0001$). **Summary.** Hospital mortality in patients with STEMI complicated by CS was associated with the presence left ventricular ejection fraction less than 40%, three-vessel coronary lesion and performing PCI later than 6 hours from the beginning of the pain attack.

Key words: *cardiogenic shock, acute ST-segment elevation coronary syndrome, hospital mortality, risk factors, predictors, prognosis*

Conflict of interests

The authors declare no conflict of interests

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CS — cardiogenic shock, ACSST — acute coronary syndrome with ST segment elevation, PCI — percutaneous coronary intervention

Due to the significant success in medical care for patients with acute coronary syndrome with ST-segment elevation (ACSST), widespread routine percutaneous coronary interventions (PCI) have reduced in-hospital mortality in recent years [1]. Today, in-hospital ACSST mortality in leading clinical centers does not exceed 2–2.3% [2]. However, despite advances in treatment, cardiogenic shock (CS) remains the leading cause of death in these patients [3]. CS accounts for 76% of lethal outcomes in myocardial infarction with ST segment elevation in the first seven days [4]. CS incidence in ACSST in recent decades has not significantly decreased and amounts to 4–15% [5, 6], and mortality is not less than 50% [7, 8]. In this regard, the identification of predictors of adverse outcome in patients with ACSST complicated by CS and the search for methods for their management that could increase the survival of these patients is relevant.

Study purpose — to study the risk factors for in-hospital mortality in patients with ACSST complicated by CS.

Material and Methods

This prospective, open-label, observational study included 104 patients with ACSST complicated by CS admitted to the Cardiology Department of the Vascular Center of Ivanovo Regional Clinical Hospital in 2019. CS was diagnosed based on systolic blood pressure decrease below 90 mm Hg for more than 30 minutes or the need for infusion of vasopressors to maintain systolic pressure above 90 mm Hg and due to the signs of hypoperfusion. Among the studied patients with CS, 58 patients died in the hospital (55.8%). Therefore, all the patients were divided into two groups. The study group I included

58 deceased patients with ACSST complicated by CS, and the comparison group (group II) included 46 patients with ACSST with CS who underwent treatment and were discharged from the hospital. All enrolled patients with ACSST at admission were diagnosed with myocardial infarction with ST segment elevation.

Inclusion criteria: ACSST complicated by CS; men and women; signing of voluntary informed consent.

Exclusion criteria: acute coronary syndrome without ST segment elevation on the electrocardiogram; intercurrent somatic disease, which has an independent negative impact on the prognosis (severe hepatocellular and respiratory failure, metastatic cancer, cerebrovascular accident during the month before or during this hospitalization); refusal of the patient to be included in the study.

Upon admission to the hospital, the study patients underwent physical examination; blood troponins, lipids, glucose, and creatinine were determined and electrocardiography and echocardiography were performed. Coronary angiography and PCI were urgently performed.

The patients were examined based on informed voluntary consent in accordance with order No. 3909n of the Ministry of Health and Social Development of the Russian Federation of April 23, 2012 (registered by the Ministry of Justice of the Russian Federation on May 05, 2012, under No. 240821) in compliance with ethical principles.

Statistical data were processed using the IBM SPSS Statistics 23 software. The normal distribution of the sample was checked using the Shapiro—Wilk test. Quantitative signs are presented as the arithmetic mean of the variation series and its standard deviation ($M \pm SD$) in the normal distribution or the median and interquartile range ($Me [Q25; Q75]$) in the distribution other than normal. Qualitative data are expressed as absolute and percentage values ($n (\%)$). Analysis of the statistical significance of differences between quantitative indicators was performed using the parametric Student's *t*-test or the non-parametric Mann—Whitney *U*-test, between the qualitative characteristics — χ^2 criterion. Factors associated with the likelihood of in-hospital mortality were identified using simple logistic regression. Multiple logistic regression was used to detect predictors that independently affect in-hospital mortality. Differences were considered significant at $p < 0.05$.

Results

The mean age of deceased patients with CS was significantly higher than that of survivors (71.8 ± 7.31 and 59.5 ± 6.18 years, respectively, $p = 0.003$) (Table 1). There were 32 (55.2%) patients older than 70 years in group I,

which is significantly higher than in group II (10 (22.7%), $p = 0.0004$). Among the studied patients with ACSST complicated by CS, men predominated, but in the group of deceased patients, there were fewer of them than among survivors (32 (55.2%) and 34 (73.9%), respectively, $p = 0.049$), due to the increased number of women (26 (44.8%) and 12 (26.1%), respectively, $p = 0.049$).

There were no significant differences between the groups in the incidence of such cardiovascular risk factors as smoking, hypertension, diabetes mellitus, obesity and dyslipidemia. Chronic kidney disease diagnosed before the development of ACSST was more common in deceased patients with CS than among survivors (32 (55.2%) and 9 (19.6%), respectively, $p = 0.0002$). The incidence of acute kidney injury was higher in the observation group than in the control group (39 (67.2%) and 22 (47.8%), respectively, $p = 0.046$). A history of myocardial infarction was observed in 30 (51.7%) patients of group I and in 9 (19.6%) patients of group II ($p = 0.001$). Prior to the development of ACSST, among deceased patients with CS, chronic heart failure with reduced ejection fraction was observed more often than among survivors (36 (62.1%) and 12 (26.1%), respectively, $p = 0.0001$); NYHA III–IV (32 (55.2%) and 11 (23.9%), respectively, $p = 0.001$). There were no significant differences between the compared groups in the frequency of PCI and cerebrovascular accident history, as well as in the localization of acute ischemic changes in the electrocardiogram.

Compared with surviving patients with CS, deceased patients with ACSST complicated by CS had significantly higher blood levels of leukocytes (13.1 ± 1.01 and $10.2 \pm 0.95 \cdot 10^9/l$, respectively, $p = 0.0001$) and troponin ($7,905.1 \pm 710.22$ and $6,134.3 \pm 811.18$ pg/ml, respectively, $p = 0.001$) (Table 2). Initially, before CAG and PCI in patients of group I, plasma creatinine was significantly higher (138.2 ± 12.12 and 116.8 ± 10.22 $\mu\text{mol/l}$, respectively, $p = 0.002$), and the glomerular filtration rate was lower (45.7 ± 4.36 and 51.3 ± 3.12 ml/min/1.73 m², respectively, $p = 0.016$) in comparison with group II. There were no significant differences between the groups in terms of lipid profile, as well as blood level of hemoglobin and glucose.

At admission, ejection fraction of the left ventricle below 40% was observed in 46 (79.3%) and 27 (58.7%) patients of group I and II, respectively ($p = 0.022$). Significant differences were found between the observation and comparison groups in terms of end-systolic (48.7 [47.9; 49.4] and 47.9 [47.3; 48.5] mm, respectively, $p = 0.013$) and end-diastolic (58.5 [57.9; 59.1] and 57.1 [56.7; 57.2] mm, respectively, $p = 0.012$) size of the left ventricle.

Urgent coronary angiography was performed in 48 (82.8%) deceased patients and in 46 (100%) survived patients with ACSST complicated by CS ($p = 0.003$).

Table 1. Initial comparative characteristics of patients with acute coronary syndrome with ST-segment elevation complicated by cardiogenic shock

Sign	Group I (n=58)	Group II (n=46)	P
Age, years (M±SD)	71,8±7,31	59,5±6,18	0,003
Age >70 years, n (%)	32 (55,2)	10 (21,7)	0,0004
Female, n (%)	26 (44,8)	12 (26,1)	0,049
Male, n (%)	32 (55,2)	34 (73,9)	0,049
Smoking, n (%)	33 (56,9)	24 (52,2)	0,635
AH, n (%)	52 (89,7)	35 (76,1)	0,064
Diabetes mellitus, n (%)	20 (34,4)	10 (21,7)	0,157
Obesity, n (%)	23 (39,7)	17 (36,9)	0,781
Dyslipidemia, n (%)	46 (79,3)	34 (73,9)	0,521
AKI, n (%)	39 (67,2)	22 (47,8)	0,046
History of CKD, n (%)	32 (55,1)	9 (19,6)	0,0002
PICS, n (%)	30 (51,7)	9 (19,6)	0,001
CHF with preserved EF, n (%)	2 (3,4)	15 (32,6)	0,00004
CHF with intermediate EF, n (%)	12 (20,7)	14 (30,4)	0,259
CHF with low EF, n (%)	36 (62,1)	12 (26,1)	0,0002
History of CVA, n (%)	4 (6,9)	6 (13,0)	0,295
History of PCI, n (%)	5 (6,9)	4 (8,7)	0,989
Localization of acute ischemic changes on the ECG:			
- anterior, n (%)	24 (41,4)	15 (32,6)	0,364
- anterolateral, n (%)	7 (12,0)	5 (10,9)	0,317
- inferior, n (%)	27 (46,6)	26 (56,5)	0,851
Time from the beginning of the pain syndrome to PCI, min (Me [Q25;Q75])	418,8 [379,1;458,6]	214,5 [171,9;257,1]	0,0001

Note: AH — arterial hypertension; AKI — acute kidney injury; CKD — chronic kidney disease; PICS — postinfarction cardiosclerosis; CHF — chronic heart failure; EF — ejection fraction; FC — functional class; CVA — acute cerebrovascular accident; PCI — percutaneous coronary intervention; ECG — electrocardiography

Table 2. Primary laboratory and instrumental indicators in patients with acute coronary syndrome with ST-segment elevation complicated by cardiogenic shock (Me [Q25;Q75])

Indicator	Group I (n=58)	Group II (n=46)	P
Leukocytes, 10 ⁹ /l	13,1 [12,1;14,1]	10,2 [9,3;11,2]	0,0001
Hemoglobin, g/l	139,8 [129,6;150,01]	143,9 [131,6;156,2]	0,124
Troponin, pg/ml	7905,1 [7194,9;8615,3]	6134,3 [5323,1;6945,5]	0,001
Creatinine, μmol/l	138,2 [126,1;150,3]	116,8 [106,6;127,02]	0,002
GFR, ml/min/1,73 m ²	45,7 [41,3;50,06]	51,3 [48,2;54,4]	0,016
Glucose, mmol/l	6,8 [5,7;7,9]	6,7 [5,7;7,7]	0,264
Total cholesterol, mmol/l	5,7 [5,3;6,1]	5,5 [5,2;5,8]	0,132
LDL, mmol/l	3,3 [3,1;3,6]	3,2 [2,9;3,5]	0,074
HDL, mmol/l	1,05 [0,96;1,14]	1,11 [0,98;1,22]	0,102
Triglycerides, mmol/l	2,3 [1,7;2,8]	2,2[1,7;2,7]	0,208
LV ejection fraction, %	33,5 [30,3;36,7]	36,4 [32,1;40,8]	0,048
LVESD, mm	48,7 [47,9;49,4]	47,9 [47,3;48,5]	0,013
LVEDD, mm	58,5 [57,9;59,1]	57,1 [56,7;57,2]	0,012

Note: GFR — glomerular filtration rate; LDL — low-density lipoproteins; HDL — high-density lipoproteins; LV — left ventricular ejection; LVESD — left ventricular end systolic diameter; LVEDD — left ventricular end diastolic diameter

CAG in the observation group was not performed in 10 (17.2%) patients due to the extremely serious state at admission and death within an hour from the moment of hospitalization.

According to the results of CAG, in patients of group I, three-vessel lesion of the coronary arteries was significantly more often diagnosed than in patients of group II (36 (75%) and 12 (26.1%), respectively, $p = 0.0001$), while single-vessel was less often (1 (2.1%) and 27 (58.1%), respectively, $p = 0.0001$). Compared with survivors, deceased patients with CS had a significantly higher average number of occlusions (1.58 [0.97; 2.19] and 1.13 [0.51; 1.75], respectively, $p = 0.001$) and hemodynamically significant stenoses (2.5 [1.75; 3.25] and 2.1 [1.48; 2.72], respectively, $p = 0.033$) of the coronary arteries. Chronic occlusion of the coronary artery unrelated to ACSST was observed in 25 (52.1%) patients of group I and 12 (26.1%) of patients of group II ($p = 0.009$). Hemodynamically significant stenosis of the left main coronary artery stem was observed in 9 (18.8%) and 2 (4.3%) patients of the observation and comparison group, respectively ($p = 0.03$).

PCI was performed in 43 (74.1%) deceased and 43 (93.5%) survived patients with ACSST with CS ($p = 0.009$). In all cases, the intervention resulted in the stenting of the infarct-related artery. The mean number of implanted stents in the observation group was 1.52 [0.81;

2.23], and in the comparison group — 1.43 [0.73; 2.13] ($p = 0.004$). In the remaining patients of group I and II (5 (10.4%) and 3 (6.5%), respectively, $p = 0.023$), stenting was not performed due to multiple coronary bed lesions and/or technical impossibility of intervention. Unsuccessful PCI was observed in 13 (30.2%) deceased and 3 (7%) survived patients with ACSST complicated by CS ($p = 0.001$) and was characterized primarily by the no-reflow phenomenon in both groups (10 (23.3%) and 3 (7%), respectively, $p = 0.007$), as well as acute stent thrombosis and death on the operating table in the observation group (2 (4.7%) and 1 (2.3%), respectively).

The mean time from the onset of pain to the intervention was greater in the group of deceased patients (418.8 [379.1; 458.6] and 214.5 [171.9; 257.1] minutes, respectively, $p = 0.0001$). The same trend was observed in the study of the frequency of PCI, performed more than six hours from the onset of angina (28 (65.1%) and 6 (14%), respectively, $p = 0.0001$).

Intraaortic balloon counterpulsation was performed in 12 (20.7%) patients of group I and in 6 (13%) patients of group II ($p = 0.301$).

Using the simple logistic regression method, we identified factors associated with in-hospital mortality in patients with ACSST complicated by CS (Table 3).

A multiple logistic regression analysis, which incrementally included the above-mentioned signs, identified

Table 3. Factors associated with hospital mortality in patients with acute coronary syndrome with ST-segment elevation complicated by cardiogenic shock

Sign	OR	95% CI	p
Age >70 years	4,43	1,85-10,59	0,001
History of CKD	5,06	2,07-12,37	0,00001
PICS	4,41	1,81-10,75	0,001
CHF III-IV FC	3,92	1,64-9,19	0,001
LV ejection fraction <40%	2,69	1,12-6,41	0,031
Three-vessel lesion	8,51	3,36-21,49	0,00001
Chronic CA occlusion	3,08	1,29-7,34	0,012
PCI later 6 hours after the onset of pain	11,51	3,96-33,44	0,00001
Unfulfilled PCI	5,02	1,35-18,53	0,01
Unsuccessful PCI	5,78	1,51-22,10	0,011

Note: OR — odds ratio; CI — confidence interval; CKD — chronic kidney disease; PICS — postinfarction cardiosclerosis; CHF — chronic heart failure; FC — functional class; LV — left ventricle; CA —coronary artery; PCI — percutaneous coronary intervention

Table 4. Independent predictors of hospital mortality in patients with acute coronary syndrome with ST-segment elevation complicated by cardiogenic shock

Sign	OR	95% CI	Wald χ^2	p
LV ejection fraction <40%	1,99	1,11-5,86	7,797	0,007
Three-vessel lesion	5,91	1,55-22,53	6,769	0,009
PCI later 6 hours after the onset of pain	3,50	1,88-13,89	8,255	0,005

Note: OR — odds ratio; CI — confidence interval; LV — left ventricle; PCI — percutaneous coronary intervention

significant independent variables that influence an adverse outcome in patients with ACSST complicated by CS (Table 4). For the model as a whole, Wald chi square was 6.676, $p < 0.01$.

Discussion

According to the literature, elderly age is an independent predictor of an adverse outcome in patients with ACSST complicated by CS, which is associated primarily with progressive left ventricular dysfunction in patients with ACS [3, 9, 10]. Age older than 65–70 years in patients with ACSST is associated with a high incidence of CS, the history of cardiovascular diseases [9] and such organizational aspect as adherence to a conservative management of older patients [10]. In the presented analysis, the majority of deceased patients with CS were older than 70 years, and the mean age in the observation group was 71.8 ± 7.3 years.

According to some authors, the female sex is a factor of in-hospital mortality in patients with CS in ACSST [9]. Other researchers noted that women with myocardial infarction complicated by CS were more likely to have unfavorable clinical characteristics such as old age, diabetes mellitus, hypertension and low cardiac output; however, the female sex was not identified as an independent predictor of in-hospital mortality in patients with CS [11, 12, 13]. According to our results, there were significantly more women in the group of deceased patients than among survivors. However, logistic regression analysis did not confirm the hypothesis that female sex may be a risk factor for in-hospital mortality in patients with ACSST complicated by CS (OR 2.3, 95% CI 0.99–5.32; $p = 0.065$).

Our findings that the clinical and anamnestic predictors of death in patients with ACSST with CS are chronic kidney disease, atherosclerosis and high-NYHA chronic heart failure are consistent with the results of other studies [3, 7, 14, 15].

In the groups we analyzed, the blood level of leukocytes and troponins on the first day after hospitalization was significantly higher in deceased patients with CS. According to the literature, a systemic inflammatory reaction plays a role in the development and progression of CS in myocardial infarction. It occurs in cardiac muscle necrosis and progressive tissue hypoxia during shock, and contributes to the progression of myocardial dysfunction [16]. The blood level of proinflammatory cytokines (interleukin-6, tumor necrosis factor- α , C-reactive protein and others) and leukocytes correlated with the severity of CS and adverse outcome in patients with myocardial infarction [16, 17]. Some studies note that the degree of blood troponin increase in patients with ACS has prognostic value in the development of CS and early mortality [18].

A high level of plasma creatinine is associated with an unfavorable prognosis in ACSST complicated by CS [3, 14]. According to our data, the initial serum creatinine was higher before PCI and the glomerular filtration rate was lower in deceased patients.

The pathogenesis of CS in myocardial infarction is based on a decrease in myocardial contractility in conditions of acute ischemia and cardiac muscle necrosis. This creates a vicious circle, that is, a decrease in cardiac output and aggravation of myocardial ischemia, which, in turn, further worsens the systolic function of the heart [16]. In this regard, it was proved that a decrease in the ejection fraction below 40% is an independent factor of fatal outcome in patients with ACSST, complicated by CS [3, 15]. This is also reflected in our study.

The analysis of coronary angiograms showed that a multiple, three-vessel lesion of the coronary bed and chronic occlusion of the coronary artery was more common in deceased patients with CS than in survived patients. A number of publications evaluate multi-vessel coronary lesion and chronic occlusion of an infarct-unrelated artery as risk factors for poor prognosis in patients with CS [16, 19]. These factors seem capable of aggravating myocardial ischemia and systolic dysfunction that already exist in patients with ACSST with CS.

According to current recommendations, primary PCI is the preferred method of reperfusion in ACSST complicated by CS [1]. Refusal of intervention or its inefficiency is associated with a high incidence of early mortality in this category of patients [10, 15]. In addition, the timing of PCI also has prognostic value. The superiority of early revascularization was proven [12, 15]: intervention within six hours after the onset of chest pain was associated with the lowest mortality in patients with CS [16]. In our study, the time between the onset of pain to PCI was significantly longer in the group of deceased patients with CS and averaged seven hours. The data obtained on the possible effect of such factors as failure to perform the intervention, unsuccessful intervention or intervention after six hours from the onset of angina on in-hospital mortality in patients with ACSST complicated by CS are consistent with the literature data. The effect of these PCI-associated predictors on in-hospital mortality in this group of patients can be associated with the volume of viable myocardium, the degree of systolic dysfunction of the left ventricle, and the severity of CS.

According to our results, multiple stenting of coronary arteries was more often performed in deceased patients with CS than in the comparison group. This observation can be explained by the severity of the coronary bed lesion, multiple stenoses and occlusions of coronary arteries in these patients, which required implantation of two or more stents for adequate myocardial reperfusion.

Conclusion

According to the study results, the risk of in-hospital mortality in patients with ACSST complicated by CS is associated with ejection fraction decrease below 40%, a three-vessel coronary lesion, and PCI after six hours from the pain onset. The identification of predictors of an unfavorable CS course can help optimize risk stratification and select the optimal management strategy for patients with ACSST in order to improve their treatment outcomes and prognosis.

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