



ACUTE HEART FAILURE AFTER MYOCARDIAL INFARCTION

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ABSTRACT

We compared two groups of patients after acute myocardial infarction. First group was treated with fibrinolytics and they were hospitalized within six hours from the beginning of the first chest-symptoms, and second group that did not come within optimal time was treated with anticoagulants only. The patients were classified according to Killip-classification, shock-index and TIMI-risk-score after myocardial infarction. Results prove great benefit of fibrinolytic therapy in optimal time, concerning both keeping myocardial muscle mass and myocardial pump function.

KEY WORDS: heart failure, Killip-classification, fibrinolytic therapy, anticoagulant therapy

INTRODUCTION

Cardiac pump failure is the leading cause of circulatory failure and in-hospital death from acute MI (myocardial infarction). Manifestations of circulatory failure may include a weak pulse, low blood pressure, cool extremities, a third heart sound, pulmonary congestion, oliguria, and cold sweat perspiration. However, several distinct mechanisms, hemodynamic patterns, and clinical syndromes characterize the spectrum of circulatory failure in acute myocardial infarction. Each requires a specific approach to diagnosis, monitoring, and therapy. The degree of left ventricular dysfunction correlates well with the extent of acute ischemia/infarction. Hemodynamic compromise becomes evident when impairment involves 20 to 25% of the left ventricle, and cardiogenic shock or death occurs with involvement left ventricular muscle of 40% or more. Pulmonary congestion and S₃ and S₄ gallops are the most common physical findings. Early recanalization (via thrombolytics, PCI, or CABG) is the most effective therapy to reduce infarct size, ventricular dysfunction, and associated heart failure. Medical treatment of heart failure related to the ventricular dysfunction of acute myocardial infarction is otherwise generally similar to that of heart failure in other setting and includes adequate oxygenation and diuresis (begun early, blood pressure permitting, and continued long-term if needed). Intravenous vasodilator therapy (for preload and after load reduction), inotropic support, and intra-aortic balloon counter pulsation are

indicated in cardiogenic shock Nitrates (nitroglycerin) reduce preload and effectively relieve congestive symptoms(1,2). Left heart weakness is linked with both long- and short-term poor prognosis. Clinical symptoms begin with dyspnea, sinus tachycardia, a third heart sound and murmurs which are first detected on the pulmonary basis but then spread and involve whole lungs. However, developed pulmonary congestion is not necessarily followed by the auscultator signs. Clinical examination (heart sound auscultation and follow-up of the rest of the vital parameters) must be periodically (repeatedly) done in all patients in the early phase of infarction. General measures include follow-up of arrhythmia, control of the electrolyte level in serum as well as registration of the collateral conditions like valvular dysfunction or lung diseases. Pulmonary congestion can be detected by the x-ray examination. Echocardiography is a very useful method in the estimation of the extent of the infarction, valvular function and appearance of mechanical complication like mitral regurgitation and ventricular septal defect, conditions that might be responsible for heart function irregularity. In patients with serious heart failure and shock, PCI or surgical revascularization can improve survival (1, 2, 3). Heart failure grade can be defined according to Killip classification (Table 1). TIMI – risk score (The Thrombolysis in Myocardial Infarction Study Group) has a higher prognostic value, especially in the estimation of one- and six-months survival (Table 2) (4). Relative or absolute hypovolemia is a frequent cause of hypotension and circulatory failure and is easily corrected if recognized and treated promptly. Poor hy-

dration, vomiting, diuresis, and disease- or drug-induced peripheral vasodilation may contribute. Hypovolemia should be identified and corrected with intravenous fluids before more aggressive therapies are considered. An empirical fluid challenge may be tried in the appropriate clinical setting (e.g., hypotension in absence of congestion; inferior or RV infarction; hypervagotonia). If filling pressures are measured, cautious fluid administration to a pulmonary capillary wedge pressure of up to about 18 mm Hg may optimize cardiac output and blood pressure without impairing oxygenation. (1) Right ventricular ischemia and infarction occur with proximal occlusion of the right coronary artery (before the take off of the right ventricular branches). Ten to fifteen percent of inferior acute ST-elevation myocardial infarctions show classic hemodynamic features, and these patients form the highest risk subgroup for morbidity and mortality (25 to 30% versus <6% hospital mortality). Improvement in right ventricular function commonly occurs over time, suggesting reversal of ischemic stunning and other favorable accommodations, if short-term management is successful. Hypotension with clear lung fields and elevated jugular venous pressure in the setting of inferior or inferoposterior acute myocardial infarction should raise the suspicion of right ventricular infarction. Kussmaul's sign (distention of the jugular vein on inspiration) is relatively specific and sensitive in this setting. Right-sided ECG leads show ST-elevation, particularly in V4R, in the first 24 hours of RV infarction. Echocardiography is helpful in confirming the diagnosis (dilation of right ventricular and dysfunction are

Class	Features	Hospital Mortality Rates (%)
1	No signs of heart failure	6
2	S3,rales,gallop,radiographic HF	17
3	Pulmonary oedema	38
4	Cardiogenic shock	81

TABLE 1. Killip classification

Risk factor	Points	Score	30-day mortality (%)
Age > 75 y	3	0	0,8
Age 65-74 y	2	1	1,6
Systolic BP < 100 mm Hg	3	2	2,2
Heart rate > 100 beats/min	2	3	4,4
Killip class > 1	2	4	7,3
Anterior MI or LBBB	1	5	12,4
DM,HTN or angina	1	6	16,1
Weight < 67 kg	1	7	23,4
Symptoms onset to treatment > 4 h	1	8	26,8
		>8	35,9

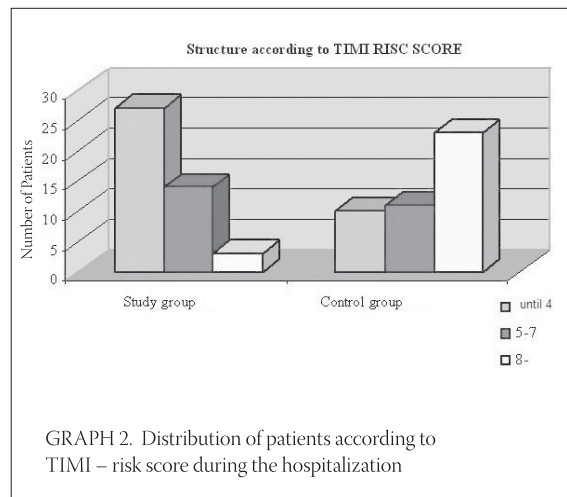
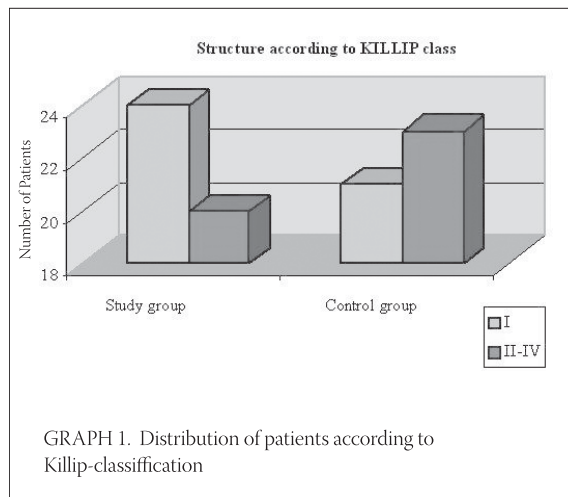
Abbreviations: BP-blood pressure; HTN-hypertension; LBBB-left bundle branch block; MI-myocardial infarction

TABLE 2. TIMI risk score for ST-segment-elevation myocardial infarction

Group	Research	Control	Total
I	24 (27,27%)	21 (23,86%)	45 (51,14%)
II-IV	20 (22,73%)	23 (26,14%)	43 (48,86%)
Total	44 (50%)	44 (50%)	88 (100%)

P=0,01

TABLE 3. Distribution of patients according to Killip-classification



TIMI– risc score	Research Group	Control Group	Total
0- 4	27 (30,68%)	10 (11,36%)	37 (42,06%)
4-7	14 (15,91%)	11 (12,5%)	25 (28,41%)
8->	3 (3,41%)	23 (25,14%)	16 (18,18%)
Total	44 (50%)	44 (50%)	88 (100%)

P=0,05

TABLE 4. Distribution of patients according to TIMI – risk score during the hospitalization

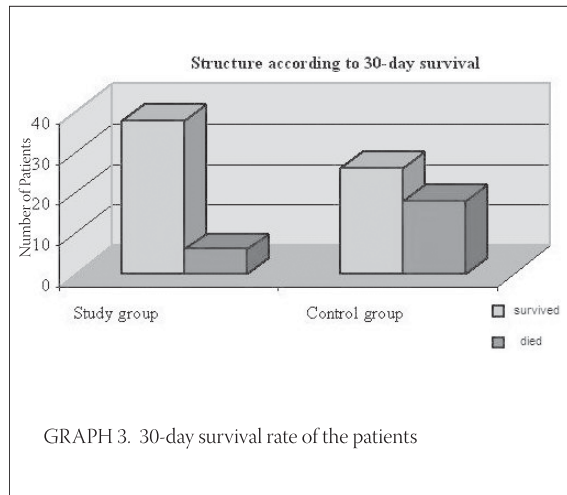
observed). If right heart pressures are measured, a right atrial pressure of ≥ 10 mmHg and $\geq 80\%$ of the pulmonary capillary wedge pressure is relatively sensitive and specific for right ventricular ischemic dysfunction (1,4). Management of right ventricular infarction consists of early maintenance of right ventricular preload, reduction of right ventricular afterload, early recanalization, short-term inotropic support if needed, and avoidance of vasodilators (e.g. nitrates) and diuretics used for left ventricular failure (which may cause marked hypotension). Volume loading with normal saline alone is often effective. If the cardiac output fails to improve after 0,5 to 1 liter of fluid, inotropic support with dobutamine is recommended. High-grade atrioventricular block is common, and restoration of atrioventricular synchrony with temporary atrioventricular sequential pacing may lead to substantial improvement in cardiac output. The onset of atrial fibrillation (in up to one third of right ventricular infarcts) may cause severe hemodynamic compromise requiring prompt cardioversion. Early coronary recanalization with thrombolysis or PCI markedly improves outcomes(1,5,6). Cardiogenic shock is

a form of severe left ventricular failure characterized by marked hypotension (systolic pressures less than 80 mm Hg) and reductions in cardiac index ($<1,8$ L/min/m²) despite high left ventricular filling pressure (pulmonary capillary wedge pressure greater than 18 mm Hg). The cause is loss of a critical functional mass ($>40\%$) of the left ventricle. Cardiogenic shock is associated with mortality rates of more than 70 to 80% despite aggressive medical therapy. Risk factors include age, large (usually anterior) acute myocardial infarction, previous myocardial infarction, and diabetes. In patients with suspected shock, hemodynamic monitoring and intra-aortic balloon counterpulsation (IABP) are indicated (5). Intubation is often necessary. With early application, urgent mechanical revascularization (PCI or CABG) affords the best chance for survival, especially in patients younger than 75 years old (6). The goal of risk stratification before and early after discharge for acute myocardial infarction is to assess ventricular and clinical function, latent ischemia, and arrhythmic risk, and to use this information for patient education and prognostic assessment and to guide therapeutic strategies (7).

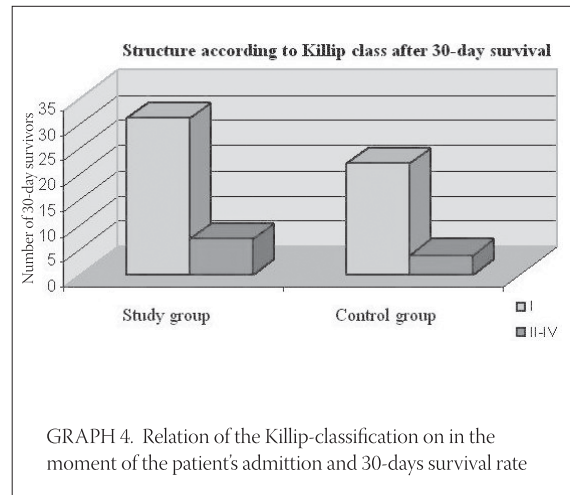
30-day survival rate of the patients	Research Group	Control Group	Total
Survived	38 (43,18%)	26 (29,54%)	64 (72,73%)
Died	6 (6,82%)	18 (20,45%)	24 (27,27%)
Total	44 (50%)	44 (50%)	88 (100%)

P=0,05

TABLE 5. 30-day survival rate of the patients



GRAPH 3. 30-day survival rate of the patients



GRAPH 4. Relation of the Killip-classification on the moment of the patient's admission and 30-days survival rate

Killip-classification	Study group of patients who survived	Control group of patients who survived	Total
I	31 (48,44%)	22 (34,37%)	53 (82,81%)
II-IV	7 (10,94%)	4 (6,25%)	11 (17,19%)
Total	38 (59,38%)	26 (40,62%)	64 (100%)

P=0,05

TABLE 6. Relation of the Killip-classification on in the moment of the patient's hospital admission and 30-days survival rate

CARDIAC CATHETERIZATION AND NONINVASIVE STRESS TESTING

Risk stratification generally involves functional assessment by one of three strategies: cardiac catheterization, submaximal exercise stress ECG before discharge (at 4 to 6 days), or symptom-limited stress testing at 2 to 6 weeks after discharge⁽¹⁾. Many patients with ST elevation-acute myocardial infarction undergo invasive evaluation for primary PCI or after thrombolytic therapy. Catheterization generally is performed during hospitalization for patients at high risk. In others, pre-discharge submaximal exercise testing (to peak heart rate of 120 to 130 beats/min or 70% of the predicted maximum) appears safe when performed in patients who are ambulating without symptoms; it should be avoided within 2 to 3 days of acute myocardial infarction and in patients with unstable post myocardial infarction angina, uncompensated heart failure, or serious cardiac arrhythmias. Alternatively or in addition, patients may undergo symptom-limited stress testing at 2 to 6 weeks before returning to work or other increased physical activities. Abnormal test results include not only ST-depression but also low functional capacity, exertional hypotension,

and serious arrhythmias. Patients with positive tests are considered for coronary angiography (8). The sensitivity of stress testing can be augmented with radionuclide perfusion imaging (thallium-201 and/or technetium-99m-sestamibi; or echocardiography. Supplemental imaging also can quantify the left ventricular ejection fraction and size the area of infarct and/or ischemia. For patients on digoxin with ST-segment changes that preclude accurate ECG interpretation (e.g., baseline LBBB or LV left ventricular hypertrophy), an imaging study is recommended with initial stress testing. In others, an imaging study may be performed selectively for those in whom the exercise ECG test is positive or equivocal. For patients unable to exercise, pharmacological stress testing can be performed using adenosine or dipyridamole scintigraphy or dobutamine echocardiography (7).

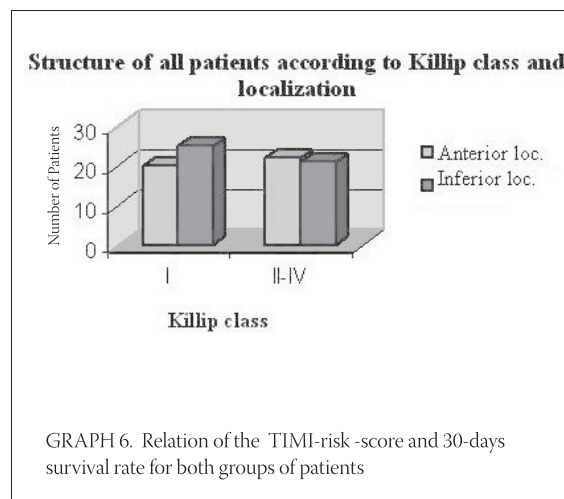
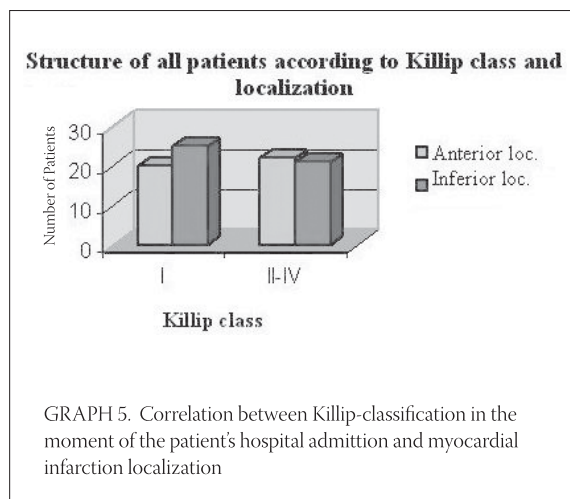
PATIENTS AND METHODS

Research included 88 patients with the myocardial infarction, with ST elevation diagnosed and treated on the Department of Internal Medicine of the General Hospital "Prim. Dr A. Nakaš", Sarajevo in

Localization	I Killip class	II-IV Killip class	Total
Anterior	20 (22,73%)	22 (25%)	42 (47,73%)
Inferior	25 (28,41%)	21 (23,86%)	46 (52,27%)
Total	45 (51,14%)	43 (48,86%)	88 (100%)

P=0,05

TABLE 7. Correlation between Killip-classification in the moment of the patient's hospital admission and myocardial infarction localization.



TIMI risc score	Survived	DOD	Total
TIMI ≤ 4	26 (29,55%)	0 (0%)	26 (29,55%)
TIMI > 4	38 (43,18%)	24 (27,27%)	62 (70,45%)
Total	64 (72,73%)	24 (27,27%)	88 (100%)

P=0,05

TABLE 8. Relation of the TIMI-risk -score and 30-days survival rate for both groups of patients

the period 01st February 2001.- 01st February 2004.

Criteria for the entering the study group were:

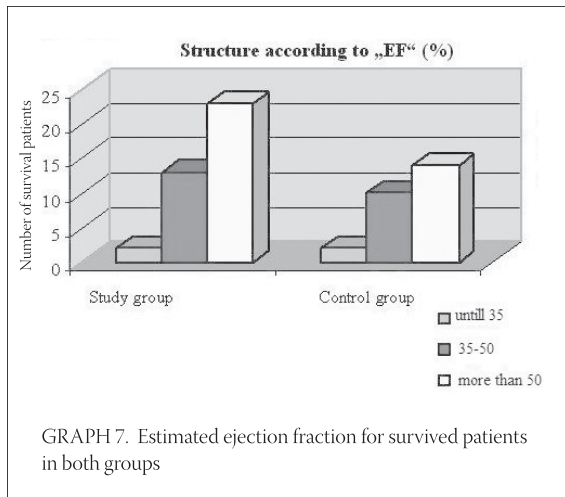
- age 41 – 71
- first myocardial infarction,
- balanced sexual distribution due to better group homogenization.

Criteria for the exclusion of patient from the study group were:

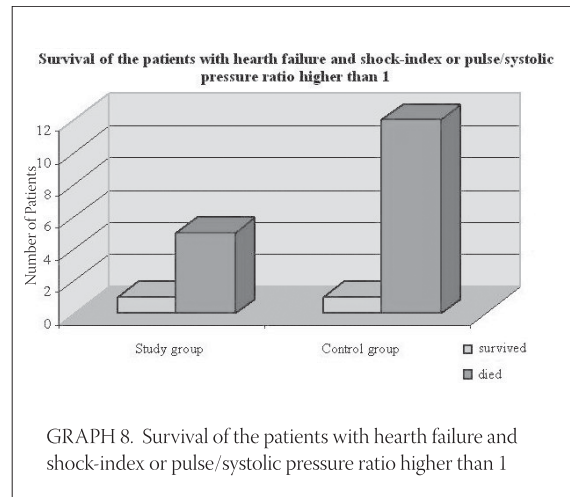
- age below 41 and above 71
- former myocardial infarction (s),

Study group had 44 patients (33 male and 11 female) admitted to the unit of intensive care with developed symptoms of acute myocardial infarction: ST elevation within 12 hours (most of them within 6 hours) from the beginning of symptoms, all treated with fibrinolytics, anticoagulants and adjuvant therapy. Control group were 44 patients (29 male and 15 female) admitted to the unit of intensive care with developed symptoms of acute myocardial infarction and ST elevation but with contraindication for fibrinolytics which therefore were not administrated. Most of the control group patients were admitted to the Hospital in the period longer

than 6 hours from the beginning of the symptoms. We divided patients in groups according to Killip-classification and parameters of the clinical presentation of the disease in the moment of hospitalization (Table 3., Graph 1.) In the first day of hospitalization according to Killip-classification there is no statistically significant difference among the groups. According to TIMI – risk score there is a statistically significant difference between the research and control group of patients (table4., graph 2). Patients that avoided fibrinolysis got additional 3 points according to TIMI - risk score that made the statistically significant difference among the groups. Analyzing of the 30-day survival rate of the research and control group we notified higher survival rate of the research group -43,18 %, compared to 29,54 % rate of the control group (table 5.,graph 3.). Statistically, in the study group survived 86% (38/48) patients and in control group 59% (26/44) patients and the survival rate after 30 days is higher in the study group for 27 %. Both groups were analyzed related to Killip classification done at the moment of patient's hospital admission and correlation of the classification results to the survival



GRAPH 7. Estimated ejection fraction for survived patients in both groups



GRAPH 8. Survival of the patients with heart failure and shock-index or pulse/systolic pressure ratio higher than 1

EF	Group	Study	Control	Total
< 35%		2 (3,125%)	2 (3,125%)	4 (6,25%)
35-50%		13 (20,31%)	10 (15,625%)	23 (35,935%)
> 50%		23 (35,94%)	14 (21,875%)	37 (57,815%)
Total		38 (59,375%)	26 (40,625%)	64 (100%)

P=0,05

TABLE 9. Estimated ejection fraction for survived patients in both groups

Survival of the patients with heart failure	Group	Study	Control	Total
Survived		1 (5,26%)	1 (5,26%)	2 (10,53%)
DOD		5 (26,32%)	12 (63,16%)	17 (89,47%)
Total		6 (31,58%)	13 (68,42%)	19 (100%)

P=0,05

TABLE 10. Heart failure incidence - shock-index or pulse/systolic pressure ratio higher than 1

rate(table 6.,graph 4.). Division of patients into the Killip classes proved to be a good predictor of one –month survival after acute myocardial infarction – STEMI.

KILLIP CLASS ON THE PATIENT HOSPITAL ADMISSION AND LOCALIZATION OF THE AMI

Both groups were analyzed related to correlation between Killip-classification in the moment of the patient’s admission and myocardial infarction localization (table 7.,graph 5.).

TIMI – RISK SCORE AND 30-DAYS SURVIVAL

Both groups were analyzed related to TIMI-risk-score and correlation of the risk-score to the 30-days survival rate(table 8.,graph 6.)

EF (%) ESTIMATION AT THE SURVIVED PATIENTS AFTER 15-30 DAYS FROM STEMI

Patients who survived divided in two groups (study and control) as defined in the study were analyzed. The aim of the analysis was to estimate whether groups of the survived patients showed significant difference regard-

ing estimated ejection fraction (EF%) (Table 9,Graph 7). Study and control group of patients after 15-30 days showed significant difference regarding estimated ejection fraction EF(%). In the study group, EF(%) was significantly higher.

Percentage relation is:

- 53 %, patients in the control group had EF over 50 %
- 60,5 % patients in the study group had EF over 50 % which makes 7,5 % higher incidence of survived EF(%) in the study group.

Study group had slightly, statistically insignificant higher survival rate of the patients with heart failure and shock-index higher than 1 compared to the control group of patients (Table 10, Graph 8)

Comment: Groups are not statistically significant. Cardiogenic shock mortality was 100 %.

DISCUSSION

During the research there were a certain variations of results related to sex and age parameters but with no statistical significance. There is also difference related

to distribution of patients according to Killip-classification which points the heart pump function. In the study group 24 patients were in Killip class I, 20 patients with Killip-class II-IV. In control group 21 patient was Killip-class I and 23 patients were Killip-class II-IV. Neither this parameter made significant statistical difference (Table 3, Graph 1) TIMI risk -score describes risk -factors and hemodynamic parameters of the patients in the moment of hospital admission in more details and proved to be useful predictor of the 30-days survival. In study group there were 27 patients with TIMI score 0-4;14 patients with TIMI-risk score 4-7 and 3 patients with score over 8. In control group there were 10 patients with TIMI score 0-4;11 patients with TIMI score 4-7 and 23 patients with score over 8. Even absolute figures show that there is a significant statistical difference between the groups (Table 4,Graph 2). This significant statistical difference between the groups according to this score is result of the fact that in the very beginning patients who did not get fibrinolytics got additional 3 points. One-month survival rate of both groups pointed out that in the study group there was a 38 survived patients after a month which makes 43,18 % from the total of all patients; in the control group 26 patients e.g. 29,54 % of total number of patients survived. Both absolute and relative calculations point the statistically significant higher number of survived patients in the study group. Mortality of the study group: $6/44 \times 100 = 13,6 \%$ Mortality of the control group $18/44 \times 100 = 40,9 \%$ Mortality in the group that got the fibrinolytics was for 27 % lower than in the control group which points the very favourising effect of fibrinolytic therapy. Study shows that fibrinolytic therapy had favourising effect on the 30-days survival of the patients with STEMI (Table 5,Graph 3). More benefits were proved at the patients with anterior localization of myocardial infarction where mortality reduction was 37% while results of our study proved the following:

- in the study group the mortality of patients with anterior localization of the myocardial infarction was 8,3 %, and in control group 46,7 %.The survival in the study group was higher for 38,4 %;
- in the study group the mortality of patients with inferior localization was 15 %, and in control group 28,5 %.The survival in the study group was higher for 13,5 %.

Killip-class of patients (in the moment of the patient's hospital admission) as predictor of the 30-days survival was compared with the survival rates as a prognostic parameter (Table 6, Graph 4). In the study group 31 of

survived 38 patients had Killip-class I, and in control group 22 of 26 survived patients had Killip-class I at the hospital admission. Killip-class I showed high statistically significant positive correlation with 30-days survival rate. Comparison of the Killip-class at the hospital admission and localization of the myocardial infarction showed the following (Table 7, Graph 5) In the group with Killip-class I at the hospital admission was approximately same number of those patients with anterior and inferior localization of myocardial infarction. Statistic analysis showed that both localization were equally presented in both groups of Killip-classification. According to the fact that anteroseptal localisation of myocardial infarction is usually more massive, it would be expected that this localization is presented with higher Killip-class, e.g. group II-IV, but we did not prove that. Possible explanation for such results could be that majority of patients who were later examined by coronarography had multivascular heart disease and were presented as first myocardial infarction both anterior or inferior localization. Detailed analysis of the risk -factors and hemodynamic parameters at the moment of patient's hospital admission is implemented in TIMI- risk -score for both group of patients.(table 8,graph 6) A fact that all patients with TIMI risk -score less or equal to 4 survived, leads to the conclusion that TIMI-scor value less or equal to 4 well correlates to the one-month survival. If less than 50% of the heart muscle mass is affected by the lesion, signs of the left ventricle heart failure are to be expected (3). The rest of the regularly perfused myocardium in the new situation tends to hypercontraction unless there is a consecutive coronary disease of it's irrigation area. Coronarography examination of the patients in this study showed that high percentage of them had multivascular heart disease which caused that incidence of the heart failure in the myocardial infarction was relatively high. More frequent heart failure at the younger population of patients can be explained with the insufficiently developed collateral circulation which could not supply the ischemic area. The worse form of the heart failure was low blood pressure appearance with the signs of the pulmonary congestion and peripheral hypoperfusion with reflex tachycardia. This situation requires prompt differentiation of cardiogenic shock versus hypovolemia or consecutive right ventricle infarction. Important parameter is CVP measurement but sometimes we are not in position to measure it although situation requires that. We should reconsider possibility of right ventricle myocardial infarction when we have infero-posterior localization of the infarction (than we have proximal occlusion of the right coronary artery), when pulsing

overloaded neck veins are obvious and when we have signs of hepatal congestion (hepatojugular reflux), with systemic hypotension, without pulmonary congestion. Analysis of the group of patients with the signs of the heart failure with the systolic pressure/puls index higher than 1 (where we registered systolic pressure below 100 mm Hg with 100 or more beats per minute- tachycardia) and the signs of the peripheral hypoperfusion. This analysis showed the following (Table 10, Graph 8):

- the study group had 6 patients with above mentioned hemodynamic parameters out of which 5 died;
- In control group there were 13 patients with 12 lethal outcomes.

Cardiogenic shock was highly lethal (almost 100 %) but similar results is registered even in the best equipped health institutions where intraaortal balloon-pump could be placed and urgent coronarography and PTCA with stent could be done according to modern protocols. In our study mortality rate of the cardiogenic shock was 100%. The only patient who survived had the consecutive infarction of the right ventricule which caused the above described hemodinamic parameters. He died 3 months after the first infarction due to development of the heavy global decompensated ischaemic cardiomyopathy. The only patient in the control group who survived was a woman who was in hypovolemia due to profuse swetting and stabilized by the volume compensation.

CONCLUSION

1. According to the results of the study fibrinolytic therapy had better effects than anticoagulant therapy in the treatment of STEMI.
2. Survival after 39-days in the study group was 86 %, compared to control group 59,1 %, and one-month survival in the study group was higher for 27 %
3. Killip-classification was tested on the complete population of patients and proved to be applicable. Killip-class I showed as favourable prognostic parameter for one-month survival and EF over 50 % after 15-30 days of STEMI
4. TIMI-risc-scor is also applicable in the study, covers the risk-factors, hemodynamic parameters and infarction localization and proved itself as 100% positive predictor of the one-month survival if equal or less than 4
5. Fibrinolytic therapy influenced mildly, statistically insignificantly survival of the complication of the heart failure in the acute myocardial infarction and mortality of the cardiogenic shock was 100 %.

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