

The clinical course of acute ST-elevation myocardial infarction in patients with hypertension

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Abstract

Background: Arterial hypertension has been documented as one of the cardiovascular risk factors. The issue whether hypertension worsens the clinical course and short-term prognosis of patients with acute ST-elevation myocardial infarction (STEMI) has been addressed by several studies, however, the results were not uniform.

Aim: To compare the clinical course and short-term prognosis in STEMI patients with or without hypertension.

Methods: The study group consisted of 366 patients with STEMI, of whom 234 (63.9%) had a history of hypertension (150 males, mean age 58.5 ± 11.2 years) whereas 132 (100 males, mean age 60.3 ± 11.9) did not. All patients underwent primary angioplasty with stent implantation. Details from medical history, cardiovascular risk factors, clinical course and in-hospital complications were recorded and compared between patients with and without hypertension.

Results: There were differences between both study groups in the prevalence of cardiovascular risk factors. Patients with hypertension had more frequently history of coronary artery disease (56 vs. 37%, $p < 0.01$), BMI $> 25 \text{ kg/m}^2$ (90 vs. 85%, $p < 0.01$), type 2 diabetes (27 vs. 14%, $p < 0.05$), hyperlipidaemia (56 vs. 43%, $p < 0.05$), and renal disease (11 vs. 5%, $p < 0.05$). Clinical course of MI was more complicated in patients with hypertension who had more often cardiogenic shock (10 vs. 6%, $p < 0.05$), pulmonary oedema (12 vs. 4%, $p < 0.05$), sinus tachycardia > 90 beats/min on admission (12 vs. 4%, $p < 0.05$), ventricular tachycardia or fibrillation (20 vs. 11%, $p < 0.01$) and complete atrioventricular block (11 vs. 4%, $p < 0.01$). In-hospital deaths occurred in 18 (7.7%) patients with hypertension and 7 (5.3%) patients without hypertension (NS). Multivariate analysis identified age > 65 years, symptoms of heart failure, atrial fibrillation, elevated blood glucose level and creatinine level as independent prognostic factors of adverse outcome in both groups whereas history of stroke, increased white cell blood count, urea level and two-vessel disease were independent prognostic variables in patients with hypertension. Ventricular tachycardia or fibrillation had prognostic significance only in STEMI patients without hypertension.

Conclusion: Patients with STEMI and hypertension have more cardiovascular risk factors and more complicated in-hospital course of MI than normotensive patients.

Key words: ST-elevation myocardial infarction, hypertension, complications

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Introduction

Acute coronary syndrome (ACS) results from pathological processes leading to the development and progression of atherosclerotic lesions in the coronary arteries, with endothelial dysfunction, increased vascular permeability, neurohormonal processes and reduced nitric oxide availability. As a consequence, penetration of oxidised LDL to the vascular smooth muscle of the media can be observed. High blood pressure (BP) is one of the major factors leading to atherogenesis and the development of vulnerable plaques which rupture, resulting in thrombosis and vessel occlusion. Long-standing hypertension adversely affects myocardial structure, and resulting myocardial

hypertrophy is associated with increased oxygen demand, leading to the development of new arterial vessels to supply the myocardium. This collateral arterial circulation, driven by BP gradient, is most effective in the subepicardial layer but much less effective in the subendocardial layer which is most prone to ischaemia and infarction. During an acute myocardial infarction (MI), BP plays a major role in the autoregulation mechanisms. Systolic BP and decreased wall tension in the ischaemic area result in patency of small vessels, providing some blood supply to the ischaemic myocardium. Large reduction of diastolic BP more often leads to ischaemia despite the lack of total vessel occlusion [1]. Numerous unique morphological and

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functional features in hypertensive patients may affect the clinical course of ACS in this population.

The aim of this study was to compare the clinical characteristics of patients with an acute ST-segment elevation MI (STEMI) with or without hypertension as well as to evaluate the clinical course in these two groups.

Methods

We evaluated 234 patients (150 men, 84 women) with STEMI and a history of hypertension (HT-STEMI) and 132 patients (100 men, 32 women) with STEMI and no history of hypertension (noHT-STEMI). These were consecutive patients admitted to our department due to an acute MI from September 2005 to May 2006. Hypertension was diagnosed based on history, clinical evaluation or previous medical documentation provided by the patients. Duration of hypertension in the HT-STEMI group ranged from 6 to 240 months (mean 90 ± 78 months). During the 2-3 months preceding the admission, BP was well controlled ($< 140/90$ mmHg) in 37 (15.8%) patients. Compared to the noHT-STEMI group, the HT-STEMI patients were more often treated with ACE inhibitors (47.4% vs. 1.5%), beta-blockers (36.3 vs. 0.8%), diuretics (16.7 vs. 0.8%) and statins (19.2 vs. 0.8%), while noHT-STEMI patients more often used long-acting nitrates (26.5 vs. 3.4%). Glycerol trinitrate use was reported by a similar proportion in both groups (52.3% of patients with noHT-STEMI and 47.4% of patients with HT-STEMI). Patients with advanced valvular disease, including aortic regurgitation (regurgitant jet area > 8 cm² and regurgitant jet volume ≥ 50 ml) and aortic stenosis (peak systolic valvular gradient > 75 mmHg and/or aortic valve area < 0.8 cm²) were excluded from the study. In all patients, the initial diagnostic work-up in the emergency department included history and physical examination, baseline electrocardiogram (ECG), BP measurement and laboratory blood tests. Patients were then transferred to the cath lab to determine the culprit vessel, assess coronary anatomy, perform angioplasty with stenting and abciximab administration, assess the immediate effect of coronary angioplasty, and determine the need for any further percutaneous coronary intervention or coronary artery bypass grafting (CABG), either emergent, urgent or elective.

The clinical course was assessed in the subsequent days, including ECG evaluation of arrhythmia, and the occurrence of major adverse cardiac events including death, stroke, and recurrent non-fatal MI. Echocardiography was performed at 5-6 day of hospitalisation. We evaluated in-hospital mortality and adverse prognostic factors.

Statistical analysis

The results are presented as mean \pm SD or numbers and percentages. The χ^2 test with or without the Yates correction, Mann-Whitney and Wilcoxon tests to compare non-normally distributed continuous variables, the Shapiro-Wilk test to verify normal distribution of continuous

variables, two-sided exact Fisher test to compare categorical variables, and the nonparametric ANOVA Kruskal-Wallis test. Uni- and multivariate logistic regression models were used to evaluate the effects of potential adverse prognostic factors in HT-STEMI and noHT-STEMI patients. For all tests, a p value < 0.05 was considered significant. Statistical analyses were performed using Microsoft Excel, STATISTICA PL 7.0 and SPSS 12.0 software. The study was approved by the local Ethics Committee.

Results

The mean age of HT-STEMI patients was 58.5 ± 11.2 years compared to 60.3 ± 11.9 years of the noHT-STEMI group ($p < 0.05$), with a similar proportion of men and women in both groups, and with 211 (57.7%) patients < 65 years of age and 155 (42.3%) patients > 65 years of age, respectively. Among patients aged < 65 years, there were 124 (33.9%) patients with HT-STEMI and 87 (23.8%) patients with noHT-STEMI (NS).

Previous diagnosis of coronary artery disease (CAD) was more common in the HT-STEMI group compared to the noHT-STEMI group (56.4 vs. 37.1%, $p < 0.01$) (Table I). Coronary artery disease risk factors, including body mass index (BMI) > 25 kg/m², hyperlipidaemia and diabetes, were significantly more common in the HT-STEMI group compared to the noHT-STEMI group (Table I). Smokers were more common among the noHT-STEMI patients. The two groups did not differ significantly with regard to the family history of CAD (Table II).

Renal diseases, including chronic glomerulonephritis and nephrolithiasis, were more common in the HT-STEMI group compared to the noHT-STEMI group ($p < 0.05$), and the prevalence of cerebrovascular disease (ischaemic stroke or transient ischaemic attack) and ultrasonographically determined atherosclerotic lesions in the carotid and lower limb arteries did not differ significantly between the two groups (Table II).

One hundred and fifty-four (65.8%) patients with HT-STEMI were admitted during the initial 6 h from the onset of pain, compared to 104 (77.3%) patients with noHT-STEMI ($p < 0.05$). Typical stenocardial pain was present in 219 (93.2%) patients with HT-STEMI and 215 (94.7%) patients with noHT-STEMI (NS).

On admission, systolic BP was 132 ± 27 mmHg in the HT-STEMI group and 123 ± 21 mmHg in the noHT-STEMI group ($p < 0.01$); diastolic BP was 80 ± 18 mmHg and 76 ± 14 mmHg, respectively ($p < 0.05$); and heart rate was 88 ± 11 bpm and 79 ± 8 bpm, respectively ($p < 0.05$).

The infarct location (Table II) and the overall number of critical coronary stenoses did not differ significantly between the two groups (Table III), with a higher proportion of critical left circumflex artery stenoses in the HT-STEMI group compared to the noHT-STEMI group (19.1 vs. 12.2%, $p < 0.05$), and a higher proportion of critical left

main coronary artery stenoses in the noHT-STEMI group compared to the HT-STEMI group (2.0 vs. 0.3%, $p < 0.05$).

Balloon angioplasty with or without stenting was performed in 261 culprit vessels in the HT-STEMI group and in 135 culprit vessels in the noHT-STEMI group. In 2 patients with noHT-STEMI, the infarct related artery patency could not be mechanically restored. Intra-aortic balloon counterpulsation (IABP) or temporary pacing during or after coronary angioplasty was required in 15 (6.4%) patients in the HT-STEMI group and 3 (2.3%) patients in the noHT-STEMI group ($p < 0.05$).

The TIMI 3 flow was restored in 217 (83.2%) patients in the HT-STEMI group and 116 patients (85.0%) in the noHT-STEMI group (NS). Further invasive treatment (second stage coronary angioplasty or CABG) was deemed necessary in 50 (21.4%) patients with HT-STEMI and 41 (32.0%) patients with noHT-STEMI ($p < 0.05$).

Cardiogenic shock occurred in 24 (10.3%) patients in the HT-STEMI group and in 8 (6.0%) patients in the noHT-STEMI group ($p < 0.05$), including on admission in 3 (1.3%) patients with HT-STEMI and 4 (3.0%) patients with noHT-STEMI ($p < 0.05$). Pulmonary oedema developed in 27 (11.5%) patients in the HT-STEMI group and 5 (3.8%) patients in the noHT-STEMI group ($p < 0.05$), including on admission in 18 (7.7%) patients with HT-STEMI and 1 (0.8%) patient with noHT-STEMI ($p < 0.01$).

Sinus tachycardia (> 90 bpm), ventricular tachycardia and/or fibrillation and third degree atrioventricular block were significantly more common in HT-STEMI patients (Table IV). Other arrhythmia and conduction disturbances (atrial tachycardia, atrial fibrillation, first and second degree atrioventricular block, His bundle branch blocks) were seen in similar proportions of HT-STEMI and noHT-STEMI patients (Table IV). Among laboratory test results, only serum urea level was significantly higher in the HT-STEMI group compared to the noHT-STEMI group (41.1 ± 22.0 vs. 34.4 ± 16.1 mg/dl, $p < 0.05$).

The duration of hospital stay was 7.0 ± 4.0 days in the HT-STEMI group compared to 7.0 ± 3.4 days in the noHT-STEMI group (NS).

Echocardiography performed at 5-6 days showed significantly reduced left ventricular ejection fraction (LVEF $< 40\%$) in 36 (15.4%) patients in the HT-STEMI group and in 28 (21.2%) patients in the noHT-STEMI group ($p < 0.05$). Twenty-five (6.8%) patients died during the hospitalisation, including 18 (7.7%) patients in the HT-STEMI group and 7 (5.3%) patients in the noHT-STEMI group (NS).

The following potential prognostic factors that might adversely affect the clinical course of STEMI were included in the analysis: age, gender, delay from the onset of pain to the performance of primary angioplasty, systolic and diastolic BP and heart rate on admission, heart failure severity, infarct location, arrhythmias and conduction disturbances, diabetes, leukocyte count, blood urea, creatinine and glucose level on admission, eccentric or

Table I. History of coronary artery disease

Parameter	HT-STEMI n = 234	noHT-STEMI n = 132	p
History of coronary artery disease, n (%)	132 (56.4)	49 (37.1)	< 0.01
Previous infarction, n (%)	51 (21.8)	30 (22.7)	NS
Previous PCI, n (%)	23 (9.8)	10 (7.6)	NS
Previous CABG, n (%)	1 (0.4)	2 (1.5)	NS

Abbreviations: PCI – percutaneous coronary intervention, CABG – coronary artery bypass graft

Table II. Coronary artery disease risk factors, concomitant diseases, and infarct location

	HT-STEMI n = 234	noHT-STEMI n = 132	p
Risk factors			
BMI ≥ 25 kg/m ² , n (%)	211 (90.2)	112 (84.8)	0.01
History of smoking, n (%)	134 (57.3)	95 (72.2)	0.01
Positive family history, n (%)	80 (34.2)	50 (37.9)	ns
Type 2 diabetes, n (%)	62 (26.5)	18 (13.6)	0.05
Hyperlipidaemia, n (%)	132 (56.4)	57 (43.2)	0.05
Concomitant diseases	n (%)	n (%)	
Renal disease	25 (10.7)	6 (4.5)	0.05
Stroke/TIA	23 (9.8)	7 (5.3)	NS
Peripheral arterial disease	11 (4.7)	7 (5.3)	NS
Carotid artery disease	11 (4.7)	5 (3.8)	NS
Infarct location	n (%)	n (%)	
Anterior/anterolateral	92 (39)	54 (40.9)	NS
Inferior/inferoposterior, right ventricle	94 (40.2)	59 (44.6)	NS
Other location	38 (21.5)	19 (14.5)	NS

Abbreviations: BMI – body mass index, TIA – transient ischaemic attack

concentric myocardial hypertrophy in echocardiography, LVEF $< 40\%$, the number of critical coronary stenoses, angioplasty failure, and the history of stroke, coronary artery disease, carotid artery disease and peripheral arterial disease.

Significant adverse prognostic factors in both HT-STEMI and noHT-STEMI groups included age > 65 years (OR 3.81, 95% CI 1.30-11.16, $p = 0.014$; and OR 5.12, 95% CI 1.10-23.75, $p = 0.036$, respectively), Killip class III or IV heart failure (OR 16.92, 95% CI 7.04-40.66, $p < 0.001$; and OR 16.27, 95% CI 3.46-76.55, $p = 0.0004$, respectively), atrial fibrillation (OR 5.10, 95% CI 1.77-14.72, $p = 0.002$; and OR 9.73, 95% CI 1.63-38.61, $p = 0.004$, respectively), blood glucose level (OR 4.89, 95% CI 1.52-15.73, $p = 0.007$; and OR 8.88, 95% CI 1.45-54.38, $p = 0.018$, respectively), and serum creatinine level (OR 9.72, 95% CI 3.87-24.39, NS; and OR 6.69, 95% CI 1.36-32.99, $p = 0.019$,

Table III. Number (percentage) of critically stenosed coronary vessels

Number of vessels	HT-STEMI n (%)	noHT-STEMI n (%)	p
One vessel	100 (42.7)	54 (31.7)	NS
Two vessels	45 (19.2)	26 (19.7)	NS
More than 2 vessels	89 (38.1)	51 (38.6)	NS

Table IV. Arrhythmias and conduction disturbances

Arrhythmias and conduction disturbances	HT-STEMI n (%)	noHT-STEMI n (%)	p
Sinus tachycardia (> 90 bpm)	27 (11.5)	5 (3.8)	0.05
Atrial tachycardia	8 (3.4)	8 (4.8)	NS
Atrial fibrillation	44 (15.2)	16 (12.2)	NS
Ventricular tachycardia and/or fibrillation	46 (19.7)	15 (11.4)	0.01
Atrioventricular block			
1 st degree	6 (2.6)	3 (2.3)	NS
2 nd degree	8 (3.4)	2 (1.5)	NS
3 rd degree	25 (10.7)	5 (3.9)	0.01
Left bundle branch block	14 (6.0)	11 (8.3)	NS
Right bundle branch block	15 (7.4)	8 (6.0)	NS

respectively). In addition, the following prognostic factors were significant only in the HT-STEMI group: history of stroke (OR 5.10, 95% CI 1.77-14.72, $p = 0.002$), leukocyte count (OR 3.02, 95% CI 1.15-7.96, $p = 0.025$), serum urea level (OR 4.48, 95% CI 1.74-11.53, $p = 0.018$) and critical two-vessel coronary disease (OR 4.73, 95% CI 1.80-12.42, $p = 0.001$). Ventricular tachycardia and/or fibrillation was a significant prognostic factor only in the noHT-STEMI group (OR 9.6, 95% CI 2.0-46.16, $p = 0.004$).

Discussion

Hypertension is one of the major risk factors of CAD and ACS. Similarly to the SYMPHONY study, [2] hypertension was present in more than half of our consecutive patients with STEMI.

Data on the effects of hypertension on in-hospital mortality are inconsistent. Rabin et al. [3] showed adverse short- and long-term outcome in hypertensive patients. In the GISSI-2 study, in-hospital and 6-month mortality in hypertensive MI patients was significantly higher compared to normotensives [4], as was the rate of left ventricular failure, recurrent angina and recurrent MI. In contrast, elevated BP was not an independent prognostic factor for 30-day mortality among MI patients in the GUSTO-1 study [5]. However, patients with very high BP were excluded from the GUSTO-1 study due to the use of thrombolytic treatment, but despite this, systolic BP

exceeded 180 mmHg in 602 patients. Ayward et al. [6] evaluated all patients participating in the GUSTO-1 study and showed that the risk of an early death was higher in patients with elevated systolic BP.

In a study by Majahalmel et al. [7], in-hospital and 6-month mortality in hypertensive and normotensive MI patients was similar, while the rate of recurrent angina, paroxysmal atrial fibrillation and acute renal failure was higher among hypertensives. Jonas et al. [8] analysed three groups of patients admitted due to MI (with normal, high normal or elevated BP) and found no significant differences in in-hospital mortality (5% among normotensives, 4% in patients with high normal BP and 1.9% among hypertensives). In our study, mortality was higher in hypertensive STEMI patients compared to normotensives (7.7 vs. 5.3%) but the difference was not significant.

The clinical course of ACS is affected by risk factors for atherosclerosis, including age, which is a significant negative prognostic factor [9-11]. Abrignani et al. [12] found higher prevalence of diabetes, dyslipidaemia, renal failure and chronic obstructive lung disease in patients with hypertension compared to normotensives. In a study by Rosengren et al. [13], the strongest risk factors in patients with ACS were hypertension, high BMI, diabetes (in men), and smoking. In our study, the prevalence of diabetes, dyslipidaemia and renal failure was higher in hypertensive STEMI patients compared to normotensives, as was higher BMI, while smokers were more common among normotensive STEMI patients.

Renal function is an important prognostic factor in hypertensive MI patients. Al-Suwaidi et al. [14] showed that reduced creatinine clearance was a significant adverse prognostic factor for mortality, including cardiovascular deaths. Anavecar et al. [15] also showed that even moderate renal dysfunction as assessed by glomerular filtration rate was associated with a higher rate of MI complications, in particular heart failure. In our study, renal function was assessed based on serum urea and creatinine level. The mean values of these parameters were significantly higher in hypertensive STEMI patients compared to normotensives.

Other adverse prognostic factors in hypertensive MI patients include neurohormonal alterations and changes in coagulation. Richard et al. [16] reported significantly higher plasma neurohormonal activity during days 1-4 and from 3-5 months following MI in hypertensives compared to normotensives. Data on the rate of haemodynamic and bioelectric complications of an acute MI in hypertensive patients are inconsistent. In a study by Abrignani et al. [12], shock, ventricular fibrillation, atrioventricular conduction disturbances, intracardiac thrombus and cardiac rupture were significantly less common in hypertensive MI patients compared to normotensives, while atrial fibrillation was more common in hypertensives. In our study, cardiogenic shock, pulmonary oedema,

ventricular tachycardia and/or fibrillation, and third degree atrioventricular block were more common in hypertensive STEMI patients compared to normotensives. In contrast, the risk of ventricular fibrillation in patients with hypertension was low in a study by Bertomeu et al. [17]. Grazuleviciene et al. [18] reported acute cardiac failure in 9.8% of MI patients. Risk factors of acute cardiac failure included hypertension and impaired glucose tolerance, similarly to a study by Wu et al. [19].

Prompt effective myocardial reperfusion results in reduced necrosis area and lower rate of MI complications [20]. Coronary flow reserve is reduced in hypertensive patients with left ventricular hypertrophy [21].

Abignani et al. [12] found no difference in the delay between the onset of pain and hospital admission between hypertensive and normotensive MI patients, while in the GREECS study, patients with hypertension presented late compared to normotensives [22]. A similar difference could be seen in our study, probably related to the higher rate of angina prior to MI in these patients, resulting in longer delay when patients with chest pain took their usual anti-anginal drugs and waited for their effect.

Infarct location and the number of critically stenosed coronary vessels were similar in our hypertensive and normotensive STEMI patients. However, coronary anatomy resulted in more hypertensive STEMI patients being referred for CABG compared to normotensives.

Drug therapy used in our hypertensive and normotensive STEMI patients was in accordance with current practice guidelines for patients with ACS. Patients with hypertension did not require additional antihypertensive drugs. This confirms observations by Gibson et al. [23], who found elevated BP ($\geq 160/100$ mmHg) in 31.7% of patients with MI or cardiac ischaemia admitted within 6 h from the onset of pain. Without the use of any antihypertensive drugs, BP remained elevated at 6 h in only 6.3% of these patients.

Conclusions

1. Hypertensive patients with STEMI have more cardiovascular risk factors compared to patients with STEMI and no history of hypertension.
2. The incidence of acute haemodynamic complications (cardiogenic shock and pulmonary oedema) requiring IABP and atrioventricular conduction disturbances requiring temporary pacing is higher in patients with hypertension.
3. In-hospital mortality tended to be higher in the HT-STEMI group, although the difference was not significant.

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Przebieg kliniczny ostrego zespołu wieńcowego z uniesieniem odcinka ST u pacjentów z nadciśnieniem tętniczym

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Streszczenie

Wstęp: Chorzy na nadciśnienie tętnicze stanowią grupę osób szczególnie predysponowanych do występowania zdarzeń sercowo-naczyniowych.

Cel: Ocena porównawcza stanu klinicznego osób z ostrymi zespołami wieńcowymi z uniesieniem odcinka ST (STEMI) chorych na nadciśnienie tętnicze i bez nadciśnienia tętniczego oraz analiza przebiegu klinicznego ostrego zespołu wieńcowego i czynników wpływających na śmiertelność wewnątrzszpitalną w obu tych grupach.

Metody: Do badań zakwalifikowano 366 chorych z ostrym zespołem wieńcowym typu STEMI, w tym z nadciśnieniem tętniczym (HT) 234 (63,9%) i bez nadciśnienia tętniczego (NT) 132 (36,1%) osoby. Wiek badanych w grupie HT-STEMI wynosił $58,5 \pm 11,2$ roku, w grupie NT-STEMI $60,3 \pm 11,9$ roku ($p < 0,05$). Czynniki ryzyka choroby wieńcowej (nadwaga i otyłość, hiperlipidemia i cukrzyca) istotnie częściej występowały u chorych z HT-STEMI, a odsetek osób palących papierosy był istotnie wyższy w grupie NT-STEMI. Choroba wieńcowa w wywiadzie częściej występowała u osób z grupy HT-STEMI aniżeli NT-STEMI (56,4 vs 37,1%, $p < 0,01$).

Wyniki: W czasie do 6 godz. od początku bólu wieńcowego rzadziej zgłaszali się chorzy z grupy HT-STEMI aniżeli NT-STEMI (65,8 vs 77,3%, $p < 0,05$). W chwili przyjęcia do szpitala skurczowe i rozkurczowe ciśnienie tętnicze oraz częstotliwość akcji serca były istotnie wyższe w grupie HT-STEMI aniżeli w grupie NT-STEMI. Elektrokardiograficzna lokalizacja zawału mięśnia sercowego oraz liczba krytycznie zwężonych naczyń wieńcowych w koronarografii nie różniły się istotnie między badanymi grupami. Chorzy byli leczeni pierwotną angioplastyką wieńcową z implantacją stentu. Kontrapulsacji wewnątrzaoortalnej lub elektrody do czasowej stymulacji podczas i po zabiegu angioplastyki wieńcowej wymagało 6,4% chorych z grupy HT-STEMI i 2,3% chorych z grupy NT-STEMI ($p < 0,05$). Wstrząs kardiogeny wystąpił u 10,3% chorych z grupy HT-STEMI i u 6,0% z NT-STEMI ($p < 0,05$), obrzęk płuc odpowiednio u 11,5 i 3,8% ($p < 0,05$). Tachykardia zatokowa ($> 90/\text{min}$), częstoskurcz komorowy i/lub migotanie komór oraz blok przedsionkowo-komorowy III stopnia istotnie częściej występowały u chorych z grupy HT-STEMI: odpowiednio 3,8, 11,4 i 3,8%, natomiast obniżoną frakcję wyrzutową lewej komory (LVEF $< 40\%$) stwierdzono u większego odsetka chorych z grupy NT-STEMI aniżeli HT-STEMI (21,2 vs 15,4%, $p < 0,05$). W czasie pobytu w klinice zmarło 25 (6,8%) chorych, w tym 18 (7,7%) w grupie HT-STEMI i 7 (5,3%) w grupie NT-STEMI. Czynniki wpływającymi na wystąpienie zgonu w obu badanych grupach były: wiek (> 65 lat), niewydolność serca powyżej II klasy wg Killipa-Kimbala, migotanie przedsionków, stężenie glukozy i kreatyniny w surowicy. Ponadto w grupie HT-STEMI udar niedokrwienny mózgu w wywiadzie, liczba leukocytów w 1 mm^3 i stężenie mocznika przy przyjęciu do szpitala, LVEF $< 40\%$ i krytyczne zwężenie dwu naczyń wieńcowych, w grupie NT-STEMI zaś dodatkowo częstoskurcz i/lub migotanie komór.

Wnioski: 1) Chorzy na nadciśnienie tętnicze (HT) ze STEMI w porównaniu z osobami bez nadciśnienia tętniczego (NT) ze STEMI są bardziej obciążeni czynnikami ryzyka miażdżycy i zdarzeń sercowo-naczyniowych. 2) Częstość ostrego powikłań hemodynamicznych (wstrząs i obrzęk płuc) wymagających kontrapulsacji wewnątrzaoortalnej oraz zaburzeń przewodzenia przedsionkowo-komorowego wymagających czasowej stymulacji jest większa u chorych z grupy HT-STEMI aniżeli u chorych z grupy NT-STEMI. 3) Śmiertelność wewnątrzszpitalna w grupie HT-STEMI jest większa aniżeli w grupie NT-STEMI, aczkolwiek nieistotnie statystycznie (7,7 vs 5,3%).

Słowa kluczowe: ostry zespół wieńcowy, nadciśnienie tętnicze, przebieg kliniczny, powikłania

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