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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The impact of renal insufficiency on in-hospital outcome in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary interventions

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Abstract

Background: Chronic renal disease (CRD) is a well-known risk factor for bleeding complications in acute coronary syndrome patients.

Aim: To determine the impact of CRD with ST segment elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (pPCI) on periprocedural complications.

Methods: 103 patients with STEMI treated with pPCI were prospectively observed for in-hospital complications and analysed according to kidney function status. Endpoints included clinical and periprocedural outcomes. Major and minor bleedings were reported according to TIMI, REPLACE2 and EASY classifications.

Results: Patients with CRD were at greater risk of major bleeding defined by RAPLACE-2 (20.0% vs. 2.7%; $p = 0.007$) and TIMI (13.3% vs. 1.3%, $p = 0.018$) classifications and had more grade 2 EASY scale haematomas (20.0% vs. 2.7%; $p = 0.007$). Vascular access crossover during PCI occurred eight-fold more often among CRD patients (33.3% vs. 4.0%, $p < 0.001$). Grade 3 TIMI flow was achieved less frequently in CRD patients (60% vs. 89.3%, $p = 0.004$). CRD predisposed to contrast-induced nephropathy (35.7% vs. 5.7%; $p < 0.001$) and ischaemic stroke (14.3% vs. 0.0%; $p = 0.004$).

Conclusions: CRD in STEMI patients undergoing pPCI is a risk factor for major and minor bleeding complications including major bleeding, moderate haematomas, contrast-induced nephropathy and ischaemic stroke. Treatment and diagnostic measures should be taken in CRD patients to reduce the severity of periprocedural complications.

Key words: chronic renal disease, percutaneous coronary intervention, bleeding complications, haemostasis, STEMI

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INTRODUCTION

Renal insufficiency is a well-defined risk factor of cardiovascular disease [1]. Patients with chronic renal disease (CRD) are diagnosed earlier and have better access to advanced medical treatment, antihypertensive drugs, antimicrobial medications, recombinant erythropoietin, calcitriol, haemodialysis, and kidney transplants. As a consequence, they survive longer [2, 3]. Despite limited evidence from randomised trials, an increasing proportion of CRD patients undergo invasive cardiovascular interventions [4]. There is evidence of increased mortality and fatal complications connected with percutaneous coronary intervention (PCI) in a CRD population [5–7]. Only a few studies have been published concerning bleeding and other clinically relevant complications in CRD patients with ST segment elevation myocardial infarction (STEMI) treated with PCI.

Theoretical background

The phenomenon of haemostasis is a complex process defined as a balance between coagulation and fibrinolysis. There is a strong correlation between CRD and haemostasis impairment. The possible pathways include: 1) platelet dysfunction

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due to retention of uraemic toxins such as hippuric, phenolic and guanidosuccinic acids [8]; and 2) hyperproduction of potent antiaggregational factor nitric oxide (previously called endothelium-derived relaxing factor) [9]. CRD is correlated with changes in the rheological properties of the blood leading to platelet dysfunction and anaemia. In patients with haematocrit below 30%, platelet adhesion is severely reduced [10]. Haemodialysis causes a sudden reduction of activated partial thromboplastic time and thrombin time, thus increasing the risk of hypercoagulation, DIC-syndrome and massive thromboembolism [11].

METHODS

The aim of this study was to compare clinical outcomes and safety of PCI in CRD patients with STEMI vs. patients with normal kidney function.

We performed a subanalysis of patients with renal failure from the OceanRace trial (Access for percutaneous coronary intervention in STEMI: radial vs. fermoral — prospective, randomised clinical trial). The inclusion criteria were: (1) pain duration between 20 min and 24 h; (2) ST segment elevation measured at the J point in two contiguous leads ≥ 0.25 mV in men below the age of 40 years, ≥ 0.2 mV in men over the age of 40 years, or ≥ 0.15 mV in women in leads V_2–V_3 and/or ≥ 0.1 mV in other leads (in the absence of left ventricular hypertrophy or left bundle branch block [LBBB]) or newly emerged LBBB; (2) age ≥ 18 years; and (3) patient's informed study consent. The study population was divided according to medical history of chronic kidney function into a CRD group and a non-CRD group. Groups were compared retrospectively.

The primary composite endpoint we defined as major bleeding complication by Randomised Evaluation in Percutaneous Coronary Intervention Linking Angiomax to Reduced Clinical Events (REPLACE)-2 and Thrombolysis In Myocardial Infarction (TIMI). Main secondary endpoint was defined as minor bleedings defined by the EASY haematoma grading for radial access and the FEMORAL haematoma grading for femoral access as presented in Table 1. In terms of clinical outcomes, the following secondary endpoints were reported: haematemesis, contrast-induced nephropathy, ischaemic stroke, length of hospital stay, sudden cardiac arrest, and death.

Vascular access is a known independent risk factor for major and minor bleeding in acute coronary syndrome patients. Partly this influence on the primary endpoint was eliminated in the present study as patients were randomised to radial or to femoral artery puncture [12]. The procedure was carried out as an urgent intervention; patients were transferred from the ambulance directly to the Cathlab, and diagnostic coronarography was performed in all cases followed by angioplasty as required. Procedural data was collected prospectively in the course of action (Table 2). Additional data was obtained from the patient's history.

Statistical analysis

Statistical measures included average and standard deviation specification for continuous data. T-test with assumption of equal variances and Pearson's χ^2 test were used respectively for continuous and nominal data analysis. Missing data was omitted.

Definitions

CRD was defined as impairment of kidney function indicated by decrease in the estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73 m^2. The eGFR was estimated by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula [13]. The post procedural coronary blood flow was evaluated by the TIMI classification, with 3 being equivalent to best flow and 0 to no-flow [14]. The pre-hospital delay was defined as time from onset of symptoms to arrival of the patient at the catheterisation room; door-to-balloon time was defined as the time from arrival at the catheterisation room to the moment the catheter guidewire crossed the culprit lesion; the procedure time was defined as the time from catheterisation room arrival to the end of the procedure. Complete definitions of bleeding classifications used in our study are presented in Table 1.

RESULTS

There were 103 patients diagnosed with STEMI. Most of the analysed patients were male (72.8%, n = 75). The average age was 60.8 ± 13.1 years. There were 16.7% (n = 15) CRD patients, who were significantly older (73.7 ± 9.5 years vs. 59.0 ± 10.7 years, $p < 0.001$) and smaller (164.3 ± 7.5 cm vs. 171.2 ± 7.9 cm, $p < 0.003$). There was no significant difference in terms of average weight, body mass index and body surface area between the two groups (Table 3).

The co-morbidities were equally spread between CRD and non-CRD groups, with no statistically significant differences: diabetes mellitus (30.8% vs. 19.4%, $p = 0.46$), hypertension (76.9% vs. 67.1%, $p = 0.746$), hyperlipidaemia (50.0% vs. 72.0%, $p = 0.124$), dysthyroidism (8.3 vs. 10.4%, $p = 1.0$), carotid artery stenosis (18.2% vs. 4.6%, $p = 0.15$), peripheral artery disease (30.0% vs. 10.8%, $p = 0.124$) and previous myocardial infarction (16.7% vs. 5.4%, $p = 0.211$). In the CRD group, there were significantly more smokers (71.8% vs. 35.7%, $p = 0.014$). On admission, patients with renal failure presented with considerably worse kidney function (eGFR: 39.54 ± 13.02 vs. 74.88 ± 32.00 mL/min/1.73 m^2, $p = 0.002$) and serum creatinine concentration (1.58 ± 0.60 vs. 0.90 ± 0.20 mg/dL, $p = 0.005$). The high-density lipoprotein concentration was significantly higher among CRD patients (51.17 ± 20.32 vs. 42.62 ±14.14 mg/dL,

Table 1. Bleeding classifications

TIMI (Thrombolysis in Myocardial Infarction) [14]	
Major	Intracranial haemorrhage
	> 5 g/dL decrease in haemoglobin concentration or > 15% absolute decrease in haematocrit
	Fatal bleeding, death within seven days
Minor	Clinically overt bleeding with > 3 g/dL decrease in haemoglobin concentration or ≥ 10% decrease in haematocrit
	No bleeding observed with ≥ 4 g/dL decrease in the haemoglobin concentration or ≥ 12% decrease in haematocrit
Minimal	Clinically overt sign of haemorrhage (including imaging) associated with a < 3 g/dL decrease in haemoglobin concentration or < 9% decrease in haematocrit
REPLACE2 (Randomized Evaluation in PCI Linking Angiomax to Reduced Clinical Events II) [27]	
Major	Intracranial haemorrhage
	Intraocular haemorrhage
	Retroperitoneal haemorrhage
	Clinically evident blood loss with a decrease in haemoglobin concentration > 3 g/dL
	Decrease in haemoglobin > 4 g/dL
	Transfusion of ≥ 2 U of whole blood or red blood cells
Minor	Any overt bleeding that does not meet criteria mentioned above
EASY (Early Discharge After Transradial Stenting of Coronary Arteries trial) local haematomas gradation [28]	
I	Local superficial haematoma of diameter below 5 cm
II	Haematoma with moderate muscular infiltration of 5–10 cm
III	Forearm haematoma with muscular infiltration larger than 10 cm below the elbow
IV	Forearm haematoma with muscular infiltration larger than 10 cm extending above the elbow
V	Haematoma with ischaemic threat (compartment syndrome)
FEMORAL local haematomas gradation [28]	
I	Local superficial haematoma of diameter below 5 cm
II	Haematoma with moderate muscular infiltration of 5–10 cm
III	Forearm haematoma with muscular infiltration larger than 10 cm above the knee and below the groin
IV	Forearm haematoma with muscular infiltration larger than 10 cm extending below the knee or above the groin
V	Haematoma ischaemic threat (compartment syndrome)

Table 2. Pharmacotherapy

	Renal failure	Without renal failure	P
Acetylsalicylic acid	16 (94.1%)	85 (97.7%)	0.419
Clopidogrel	17 (100.0%)	85 (97.7%)	0.528
Unfractionated heparin	16 (94.1%)	84 (96.6%)	0.633
Low-molecular-weight heparin	0 (0.0%)	1 (1.3%)	0.653
IIb/IIIa antagonists	9 (69.2%)	43 (62.3%)	0.635
ReoPro (abciximab)	6 (46.2%)	33 (48.5%)	0.875
Integrillin (eptifibatide)	3 (23.1%)	9 (13.0%)	0.348

p = 0.040); other fractions of cholesterol did not differ between groups. Full details are provided in Table 4.

The adherence to guidelines on anticoagulation and antiplatelet drugs was very good, 97.2% of patients received acetylsalicylic acid, 98% clopidogrel and 96.1% unfractionated heparin. Glycoprotein IIb/IIIa inhibitors were given in 62.8% of patients upon operators' discretion. There were no major differences in terms of pre- and periprocedural pharmacotherapy between the study groups — details are presented in Table 2.

The coronary angiogram revealed some differences in the angiographic presentation of CRD patients. The left artery descending and posterior descending artery were occluded considerably more often among patients with renal failure

Table 3. Baseline and demographic characteristics

	Renal failure	Without renal failure	P
General characteristics			
Age [years]	73.7 ± 9.5	59.05 ± 10.7	0.001
Male	12 (80.0%)	56 (76.7%)	0.782
Height [cm]	164 ± 8	171 ± 8	0.003
Weight [kg]	71.7 ± 14.2	77.7 ± 14.8	0.158
Body mass index	26.60 ± 5.32	25.92 ± 5.01	0.635
Body surface area [m^2]	1.80 ± 0.19	1.90 ± 0.22	0.103
Past medical history			
Diabetes mellitus	4 (30.8%)	14 (19.4%)	0.460
Hypertension	10 (76.9%)	47 (67.1%)	0.746
Hyperlipidaemia	7 (50.0%)	54 (72.0%)	0.124
Hypo- or hyperthyroidism	1 (8.3%)	7 (10.4%)	0.823
Carotid stenosis	2 (18.2%)	3 (4.6%)	0.15
Peripheral artery disease	3 (30.0%)	7 (10.8%)	0.124
Smoker	5 (35.7%)	51 (71.8%)	0.014
Previous myocardial infarction	2 (16.7%)	3 (5.4%)	0.211

Table 4. Hospitalisation characteristics

	Renal failure	Without renal failure	P
Admission data			
Pre-hospital delay [h]	4.70 ± 4.32	5.06 ± 4.46	0.749
Heart rate [bpm]	75 ± 25	80 ± 18	0.348
Systolic blood pressure [mm Hg]	127 ± 36	139 ± 27	0.101
Diastolic blood pressure [mm Hg]	67 ± 20	76 ± 15	0.055
Platelet count [K/μL]	217.6 ± 73.78	229.0 ± 61.93	0.591
Serum creatinine [mg/dL]	1.59 ± 0.60	0.90 ± 0.20	< 0.0001
Estimated glomerular filtration rate [mL/min/1.73 m^2]	39.5 ± 13.02	74.8 ± 32.00	< 0.0001
Total cholesterol levels [mg/dL]	183.2 ± 51.97	199.2 ± 45.39	0.186
LDL cholesterol levels [mg/dL]	112.8 ± 43.25	123.7 ± 40.15	0.303
HDL cholesterol levels [mg/dL]	51.1 ± 20.32	42.6 ± 14.14	0.040
Triglycerides levels [mg/dL]	120.0 ± 79.66	169.6 ± 130.69	0.172
Post-discharge summary			
Duration of hospitalisation [days]	8.8 ± 5.6	7.8 ± 5.9	0.557
Change in haemoglobin concentration [mg/dL]	–1.43 ± 1.60	–0.93 ± 1.35	0.195
Change in platelet concentration [K/μL]	–35.88 ± 25.41	–25.41 ± 38.64	0.368
Change in plasma creatinine [mg/dL]	+0.29 ± 0.84	–0.93 ± 0.20	0.005

than among the rest of the study group (66.7% vs. 40.0%, p = 0.05; 6.7% vs. 0.0%, p = 0.04). The frequency of occlusion of the remaining vessels did not differ.

The vascular access crossover rate was eight-fold higher among renal patients (33.3% vs. 4.0%, p < 0.001). The pre-hospital delay, door-to-balloon time and the procedure time were equivalent in both groups, with a mean of 4.8 ± 4.26 h, 24.7 ± 13.46 min, and 46.5 ± 22.98 min, respectively. Stents were implanted equally frequently in both groups (86.7% vs. 89.3%, p = 0.671). No drug eluting stents were implanted in the CRD group (0% vs. 14.5%, p = 0.20). Renal failure predisposed towards worse blood flow post implantation, and fewer CRD patients had TIMI 3 flow (60% vs. 89.3%, p = 0.01). Exact TIMI flow data is presented in Table 5.

Table 5. Percutaneous coronary intervention characteristics and outcomes

	Renal failure	Without renal failure	P
Infarct related artery			
Left main	0 (0.00%)	1 (1.3%)	0.653
Left anterior descending	10 (66.7%)	30 (40.0%)	0.051
Diagonal branches	0 (0.00%)	1 (1.3%)	0.653
Left circumflex	0 (0.0%)	5 (6.7%)	0.585
Marginal branches	0 (0.00%)	2 (2.7%)	0.522
Right coronary artery	4 (26.7%)	32 (42.7%)	0.248
Posterior descending artery	1 (6.7%)	0 (0.00%)	0.42
Postero-lateral artery	0 (0.00%)	2 (2.7%)	0.522
Vascular access			
Radial access	10 (58.8%)	44 (50.6%)	0.534
Femoral access	7 (41.2%)	43 (49.4%)	0.534
Vascular access crossover	5 (33.3%)	3 (4.0%)	< 0.001
Number of stents implanted			
0	2 (13.3%)	8 (10.7%)	0.556
1	9 (60.0%)	52 (71.2%)	0.285
2	4 (26.7%)	11 (15.1%)	0.230
3	0 (0.0%)	2 (2.7%)	0.693
Bare metal stent	12 (85.7%)	57 (77.0%)	0.517
Drug-eluting stent	0 (0.00%)	9 (12.2%)	0.118
No stent implantation (POBA)	2 (14.3%)	8 (10.8%)	0.379
Postoperative flow			
TIMI 0	1 (6.7%)	3 (4.0%)	0.647
TIMI 1	0 (0%)	0 (0%)	1.0
TIMI 2	4 (26.7%)	1 (1.3%)	< 0.001
TIMI 3	9 (60%)	67 (89.3%)	0.004
Door-to-balloon time [min]	27.39 ± 13.76	24.16 ± 12.72	0.379
Procedure time [min]	52.64 ± 21.07	45.57 ± 2.07	0.257
Radiation time [min]	13.86 ± 4.76	12.67 ± 8.62	0.606
Injected contrast [mL]	198.00 ± 67.00	188.78 ± 86.11	0.697

POBA — plain old balloon angioplasty; TIMI — Thrombolysis In Myocardial Infarction

CRD patients reached the primary endpoint defined (major bleeding defined by REPLACE-2 and TIMI classifications) more often than the non-CRD group (20% vs. 2.7%, p = 0.03 and 13.3% vs. 1.3%, p = 0.018). In the CRD group, there were also more subcutaneous bruises of 5–10 cm in the CRD group reported (20.0% vs. 2.7%, p = 0.007). The composite minor bleeding endpoint did not show any differences. TIMI minor bleeding was observed in 33.3% vs. 12.0% (p = 0.07) and TIMI minimal in 13.3% vs. 13.3% (p = 1.00).

Patients with renal failure were at higher risk of contrast-induced nephropathy (35.7% vs. 5.7%, p = 0.005) and ischaemic stroke (14.3% vs. 0.0%, p = 0.037). There was a trend towards a higher rate of in-hospital mortality (15.4% vs. 1.7%, p = 0.084) and sudden cardiac arrest (16.7% vs. 1.7%, p = 0.074) among CRD patients. Complete data on complications is provided in Table 6.

DISCUSSION

Main findings

We have shown that the presence of renal failure is related to a higher prevalence of periprocedural PCI complications including: major and minor bleedings, need for vascular access crossover, poor coronary flow post PCI, contrast induced nephropathy, and ischaemic stroke.

Interpretation of the results

Our findings are consistent with those from the studies conducted by Rubinstein et al. [5], Best et al. [6] and Reinecke et

Table 6. Complications

	Renal failure	Without renal failure	P
Bleeding complications			
Major bleeding complications			
Major bleeding (REPLACE2 scale)	3 (20.0%)	2 (2.7%)	0.031
Major bleeding (TIMI scale)	2 (13.3%)	1 (1.3%)	0.018
Minor bleeding complications			
EASY I	2 (13.3%)	10 (13.3%)	1.00
EASY II	3 (20%)	2 (2.7%)	0.007
EASY III	0 (0.00%)	5 (6.7%)	0.303
EASY IV	1 (6.7%)	2 (2.7%)	0.431
Overall minor bleeding (any EASY grade)	6 (40.0%)	19 (25.3%)	0.247
Minor bleeding (TIMI scale)	5 (33.3%)	9 (12.0%)	0.070
Minimal bleeding (TIMI scale)	2 (13.3%)	10 (13.3%)	1.000
Minor bleeding (REPLACE2 scale)	6 (40.0%)	19 (25.3%)	0.247
Other clinical complications			
Haematemesis	1 (6.7%)	0 (0.0%)	0.167
Contrast-induced nephropathy	4 (5.7%)	5 (35.7%)	0.005
Ischaemic stroke	2 (14.3%)	0 (0.00%)	0.037
In-hospital death	2 (15.4%)	1 (1.7%)	0.084

al. [7] who investigated the relation between renal failure and postprocedural complications. As previously shown, major bleeding is correlated with the risk of death and long-term complications [15]. In our study, the tendency of bleeding complications was noticeably higher in the CRD group, which could indirectly suggest that renal patients are potentially at increased risk of death after PCI. Conversely, this population is at increased risk of coronary ischaemia, plaque rapture and acute coronary syndrome, therefore the net clinical benefit remains in favour of PCI. The pathomechanisms behind higher ischaemic risk in CRD patients include the influence of uraemic toxins, hypertension and secondary hyperparathyroidism on the epithelium [10] and formation of wall lesions, calcifications and secondary stenosis of the coronary vessels [16]. Decreased kidney function is a known risk factor for gastrointestinal bleeding, haematemesis, contrast-induced nephropathy, sudden cardiac arrest and death [5–7, 17]. The correlation of CRD and major bleeding or death has also been confirmed in STEMI patients treated with primary PCI [18–20].

Renal related vasculopathy, small vessel diameter and extensive wall calcifications explain to some extent the observed increased crossover rate among CRD patients. Osten et al. [21], based on data from 10,821 unselected PCI patients, reported that CRD does correlate with poor procedural outcomes represented by worse residual stenosis, larger number of undeliverable stents and smaller stent diameter. In our study, we also found a trend towards lower angiographic success rate among CRD patients. The long-term data shows that presence of chronic renal insufficiency at the time of PCI for acute coronary syndrome is an independent factor for ten-year all-cause mortality (hazard ratio [HR]: 2.31, 95% confidence interval [CI] 1.25–4.29, $p = 0.008$) and cardiovascular mortality (HR 3.76, 95% CI 1.60–8.80, $p = 0.002$) [22].

As previously reported, in CRD there are more perfusion disturbances and a greater reduction of coronary blood flow. This finding is not related to the epicardial coronary artery stenosis but rather indicates microvascular abnormalities [23, 24]. We have observed similar outcomes in our cohort of patients: the post-procedural TIMI flow was decreased in the CRD group.

Patient's kidney function must be considered when choosing the type of coronary stent in patients undergoing coronary intervention. Resmini et al. [25] presented data on 219 patients with creatinine clearance below 60 mL/min/1.73 m^2 treated with PCI and bare metal stent (BMS) or drug eluting stent (DES) implantation. The incidence of death, myocardial infarction, repeated PCI or stent thrombosis was significantly higher after 48 months in those who received BMS (71% vs. 38%, $p < 0.001$). Similar findings were reported in a large retrospective analysis of 121,446 patients above 65 years of age. In a 30-month follow-up, patients with DES had lower risks of revascularisation (HR 0.91, 95% CI 0.86–0.95), myocardial infarction (HR 0.77, 95% CI 0.71––0.83), and death (HR 0.73, 95% CI 0.69–0.77) [19]. Contrary to these retrospective findings, post hoc analysis from the large randomised HORIZON-AMI trial (Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarc-

tion) revealed no advantage of DES over BMS implantation in CRD patients in terms of risk of death, reinfarction, target vessel revascularisation (TVR) for ischaemia, or stroke (33.3% vs. 33.6%, p = 0.9), and there was no difference in rates of TVR in BMS vs. DES at three years (14.1% vs. 15.1%, p = 0.8) [18]. Analysis of data from our study did not reveal any difference between DES and BMS groups, although the lack of statistical significance might be due to the limited number of individuals in the study.

Vuurmans et al. [26] presented data of 69,214 patients from the British Columbia Cardiac Registry who underwent PCI. The type of vascular route had an impact on the risk of postprocedural kidney injury. Radial access was associated with a lower risk of developing dialysis dependency (0.2% vs. 0.4%, p < 0.0001), and risk of stage 4 or 5 chronic kidney disease (0.1% vs. 0.4%, p < 0.0001).

Limitations of the study

The main limitation of our study is the low number of patients, which may hinder clinical differences. We were able to perform analysis only within the renal failure population. This was a post hoc analysis; therefore the results must be interpreted with caution.

CONCLUSIONS

Renal failure remains an important risk factor for complications related to cardiovascular interventions. Physicians must take extra care and safety measures during the periprocedural period. A low threshold for recognition of bleeding complications is advised.

Conflict of interest: *none declared*

References

1. Stenvinkel P, Heimburger O, Paultre F et al. Strong association between malnutrition, inflammation, and atherosclerosis in chronic renal failure. Kidney Int, 1999; 55: 1899–1911.
2. Hill CJ, Fogarty DG. Changing trends in end-stage renal disease due to diabetes in the United kingdom. J Renal Care, 2012; 38 (suppl. 1): 12–22.
3. Chan R, Michelis MF. Renal failure: why today's patients live better and longer. Geriatrics, 1996; 51: 37–40, 43.
4. Yu J, Ooi SY, Sergie Z, Baber U. Is there attenuation of benefit of invasive therapy in patients with chronic kidney disease? Results from randomized trials and registry data. Curr Cardiol Reports, 2012; 14: 521–527.
5. Rubenstein MH, Harrell LC, Sheynberg BV et al. Are patients with renal failure good candidates for percutaneous coronary revascularization in the new device era? Circulation, 2000; 102: 2966–2972.
6. Best PJ, Lennon R, Ting HH et al. The impact of renal insufficiency on clinical outcomes in patients undergoing percutaneous coronary interventions. Journal of the Am Coll Cardiol, 2002; 39: 1113–1119.
7. Reinecke H, Schaefer RM. Percutaneous coronary interventions in patients with mild to moderate chronic renal failure: to dilate or not to dilate? Nephrology, dialysis, transplantation: official publication of the European Dialysis and Transplant Association. European Renal Association, 2003; 18: 2218–2221.
8. Rabelink TJ, Zwaginga JJ, Koomans HA, Sixma JJ. Thrombosis and hemostasis in renal disease. Kidney Int, 1994; 46: 287–296.
9. Noris M, Benigni A, Boccardo P et al. Enhanced nitric oxide synthesis in uremia: implications for platelet dysfunction and dialysis hypotension. Kidney Int, 1993; 44: 445–450.
10. Eberst ME, Berkowitz LR. Hemostasis in renal disease: pathophysiology and management. Am J Med, 1994; 96: 168–179.
11. Surovikina MS, Samoilenko VV, Vlasova EA et al. [Changes in plasmic and platelet hemostasis in patients with chronic renal failure in hemodialysis]. Urologiia, 2012: 25–29.
12. Genereux P, Mehran R, Palmerini T et al. Radial access in patients with ST-segment elevation myocardial infarction undergoing primary angioplasty in acute myocardial infarction: the HORIZONS-AMI trial. Journal of EuroPCR in collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology. EuroIntervention, 2011; 7: 905–916.
13. Levey AS, Stevens LA, Schmid CH et al., A new equation to estimate glomerular filtration rate. Ann Int Med, 2009; 150: 604–612.
14. Chesebro JH, Knatterud G, Roberts R et al. Thrombolysis in Myocardial Infarction (TIMI) Trial, Phase I: A comparison between intravenous tissue plasminogen activator and intravenous streptokinase. Clinical findings through hospital discharge. Circulation, 1987; 76: 142–154.
15. Mrdovic I, Savic L, Asanin M et al. Sex-Related Analysis of Short- and Long-term Clinical Outcomes and Bleeding Among Patients Treated With Primary Percutaneous Coronary Intervention: an evaluation of the RISK-PCI data. Can J Cardiol, 2013; 29: 1097–1103.
16. Covic A, Kanbay M, Voroneanu L et al. Vascular calcification in chronic kidney disease. Clinical Science, 2010; 119: 111–121.
17. Herzig SJ, Rothberg MB, Feinbloom DB et al. Risk Factors for Nosocomial Gastrointestinal Bleeding and Use of Acid-Suppressive Medication in Non-Critically Ill Patients. J Gen Intern Med, 2013; 28: 683–690.
18. Saltzman AJ, Stone GW, Claessen BE et al. Long-term impact of chronic kidney disease in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention: the HORIZONS-AMI (Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarction) trial. J Am Coll Cardiol Cardiovasc Int, 2011; 4: 1011–1019.
19. Tsai TT, Messenger JC, Brennan JM et al. Safety and efficacy of drug-eluting stents in older patients with chronic kidney disease: a report from the linked CathPCI Registry-CMS claims database. J Am Coll Cardiol, 2011; 58: 1859–1869.
20. Fox CS, Muntner P, Chen AY et al. Use of evidence-based therapies in short-term outcomes of ST-segment elevation myocardial infarction and non-ST-segment elevation myocardial infarction in patients with chronic kidney disease: a report from the National Cardiovascular Data Acute Coronary Treatment and Intervention Outcomes Network registry. Circulation, 2010; 121: 357–365.
21. Osten MD, Ivanov J, Eichhofer J et al. Impact of renal insufficiency on angiographic, procedural, and in-hospital outcomes following percutaneous coronary intervention. Am J Cardiol, 2008; 101: 780–785.
22. Dohi T, Kasai T, Miyauchi K et al. Prognostic impact of chronic kidney disease on 10-year clinical outcomes among patients with acute coronary syndrome. J Cardiol, 2012; 60: 438–442.
23. Sobkowicz B, Tomaszuk-Kazberuk A, Kralisz P et al. Coronary blood flow in patients with end-stage renal disease assessed by thrombolysis in myocardial infarction frame count method. Nephrology, dialysis, transplantation: official publication of the European Dialysis and Transplant Association, European Renal Association, 2010; 25: 926–930.
24. Fineschi M, Bravi A, Gori T. The "slow coronary flow" phenomenon: evidence of preserved coronary flow reserve despite

increased resting microvascular resistances. Int J Cardiol, 2008; 127: 358–361.
25. Resmini C, Di Cuia M, Ballocca F et al. Short and long term outcome of percutaneous coronary intervention with drug eluting stent and bare metal stent in patients with chronic kidney disease. Minerva Cardioangiologica, 2012; 60: 573–580.
26. Vuurmans T, Byrne J, Fretz E et al. Chronic kidney injury in patients after cardiac catheterisation or percutaneous coronary intervention: a comparison of radial and femoral approaches (from the British Columbia Cardiac and Renal Registries). Heart, 2010; 96: 1538–1542.
27. Lincoff AM, Bittl JA, Harrington RA et al. Bivalirudin and provisional glycoprotein IIb/IIIa blockade compared with heparin and planned glycoprotein IIb/IIIa blockade during percutaneous coronary intervention: REPLACE-2 randomized trial. JAMA, 2003; 289: 853–863.
28. Bertrand, O.F, De Larochellière R, Rodés-Cabau J et al. A randomized study comparing same-day home discharge and abciximab bolus only to overnight hospitalization and abciximab bolus and infusion after transradial coronary stent implantation. Circulation, 2006; 114: 2636–2643.

Ocena kliniczna chorych z niewydolnością nerek leczonych pierwotną angioplastyką wieńcową z powodu zawału serca z uniesieniem odcinka ST

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Streszczenie

Wstęp: Przewlekła choroba nerek jest istotnym czynnikiem ryzyka powikłań krwotocznych u chorych z ostrym zespołem wieńcowym.

Cel: Celem badania była ocena wpływu przewlekłej choroby nerek na powikłania u chorych z zawałem serca z uniesieniem odcinka ST (STEMI) leczonych pierwotną angioplastyką wieńcową (pPCI).

Metody: Prospektywnej obserwacji wewnątrzszpitalnej poddano 103 chorych ze STEMI leczonych pPCI. Badanymi punktami końcowymi były powikłania okołozabiegowe i wewnątrzszpitalne, w tym m.in. duże i małe krwawienia (skale TIMI, REPLACE2 i EASY).

Wyniki: U pacjentów z przewlekłą chorobą nerek stwierdzono podwyższone ryzyko dużych krwawień ocenianych wg skali REPLACE2 (20,0% *vs*. 2,7%; p = 0,007) oraz TIMI (13,3% *vs*. 1,3%; p = 0,018) oraz większe ryzyko krwawienia w miejscu dostępu w stopniu 2 wg klasyfikacji EASY (20,0% *vs*. 2,7%; p = 0,007). Konieczność zmiany miejsca dostępu była 8-krotnie częstsza w grupie osób z przewlekłą chorobą nerek (33,3% *vs*. 4,0%; p < 0,001). Przepływ TIMI 3 po zabiegu uzyskiwano istotnie rzadziej u pacjentów z przewlekłą chorobą nerek (60% *vs*. 89,3%; p = 0,004). Przewlekła choroba nerek predysponowała do wystąpienia nefropatii kontrastowej niewydolności nerek (35,7% *vs*. 5,7%; p < 0,001) oraz udaru niedokrwiennego (14,3% *vs*. 0,0%; p = 0,004).

Wnioski: Przewlekła choroba nerek u osób ze STEMI leczonych pPCI jest istotnym czynnikiem ryzyka powikłań krwotocznych, nefropatii kontrastowej niewydolności nerek i udaru mózgu w okresie wewnątrzszpitalnym. Wybór metody leczenia powinien uwzględniać redukcję powikłań i wczesne ich rozpoznawanie.

Słowa kluczowe: przewlekła choroba nerek, angioplastyka wieńcowa, powikłania krwotoczne, krwiaki, zawał serca z uniesieniem odcinka ST

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