Kardiologia Polska 2015; 73, 7: 520–526; DOI: 10.5603/KP.a2015.0054 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Platelet count and volume indices in patients with contrast-induced acute kidney injury and acute myocardial infarction treated invasively

Paweł Francuz[1], Jacek Kowalczyk[1], Ryszard Swoboda[2], Katarzyna Przybylska-Siedlecka[1], Monika Kozieł[1], Tomasz Podolecki[1], Andrzej Świątkowski[1], Radosław Lenarczyk[1], Beata Średniawa[1], Zbigniew Kalarus[1]

[1]Department of Cardiology, Congenital Heart Diseases, and Electrotherapy, Medical University of Silesia, Silesian Centre for Heart Diseases, Zabrze, Poland

[2]Medical University of Silesia, Katowice, Poland

Abstract

Background: The aetiology of contrast-induced acute kidney injury (CI-AKI) is not well understood. We hypothesised that the pathophysiology of CI-AKI and impaired coronary reperfusion (IR), observed after invasive treatment of acute myocardial infarction (AMI), could be similar and might be related to platelet count (PC) and platelet volume indices (PVI).

Aim: To evaluate the relation between PC, PVI, IR, and CI-AKI in patients with AMI treated invasively.

Methods: A single-centre study evaluated 607 consecutive AMI-patients treated invasively. Comparative analyses were performed between patients with CI-AKI and without CI-AKI for the total study population (CI-AKI, n = 156; 25.7% vs. nCI-AKI, n = 451; 74.3%), for patients with diabetes mellitus (CI-AKI-DM, n = 56; 9.2% vs. nCI-AKI-DM, n = 123; 20.3%), and for patients with baseline kidney dysfunction (CI-AKI-BKD, n = 31; 5.1% vs. nCI-AKI-BKD, n = 67; 11.0%). Subjects with IR, who developed CI-AKI, were compared to the remaining patients with respect to platelet parameters (CI-AKI-IR, n = 47; 7.7% vs. controls, n = 560; 92.3%). For total population, as well as studied subgroups, multivariate logistic regression analyses were performed to reveal independent factors associated with CI-AKI. The results of the models were reported as odds ratios (OR) and 95% confidence intervals (95% CI).

Results: PC was higher in CI-AKI-DM-patients (224.8 ± 62.8 × 10^9/L vs. 197.9 ± 63.3 × 10^9/L; p = 0.014) and in CI-AKI-BKD-patients (248.9 ± 86.5 × 10^9/L vs. 202.5 ± 59.3 × 10^9/L; p = 0.004) than in appropriate controls. Within the studied groups, there were no differences between CI-AKI and nCI-AKI patients with respect to PVI. Comparing CI-AKI-IR-patients with controls, no differences in PC or PVI were found. IR was observed more often in CI-AKI-patients than in nCI-AKI-patients only among diabetics (48.2% vs. 27.6%; p = 0.008). Increase in admission PC was independently associated with CI-AKI in patients with diabetes (per one unit increase OR 1.006; CI 1.0–1.01; p = 0.04) as well as with baseline kidney dysfunction (per one unit increase OR 1.01; CI 1,0–1,02; p = 0.02).

Conclusions: Any similarities in the pathophysiology of CI-AKI and IR were not reflected in platelet parameters. CI-AKI development was not related to PVI; however, higher PC was an independent risk factor for CI-AKI in patients with diabetes or baseline kidney dysfunction.

Key words: acute myocardial infarction, contrast-induced acute kidney injury, diabetes mellitus, platelet count, platelet volume indices

Kardiol Pol 2015; 73, 7: 520–526

INTRODUCTION

Contrast-induced acute kidney injury (CI-AKI) or contrast-induced nephropathy (CIN) occurrence is associated with worse prognosis in patients after acute myocardial infarction (AMI). However, the pathophysiology of the disease is still not known and no completely successful prevention or treatment has been

Address for correspondence:
Paweł Francuz, MD, Department of Cardiology, Congenital Heart Diseases, and Electrotherapy, Medical University of Silesia, Silesian Centre for Heart Diseases, ul. Curie-Sklodowskiej 9, 41–800 Zabrze, Poland, e-mail: pawel.francuz@wp.pl

Received: 12.09.2014 **Accepted:** 22.01.2015 **Available as AoP:** 06.03.2015

Copyright © Polskie Towarzystwo Kardiologiczne

proposed. The potential cause of CI-AKI is temporary renal vasoconstriction with subsequent return of blood flow and reperfusion injury [1]. Similarly, one of the types of reperfusion injury is the "no-reflow" or "slow flow" phenomenon associated with percutaneous coronary interventions (PCI). Platelets have a crucial role in acute coronary syndromes (ACS) and might be implicated in no-reflow through microvascular obstruction by platelet aggregates and release of platelet-derived vasoactive and chemotactic mediators [2–4]. It has been demonstrated that high mean platelet volume was an independent predictor of impaired coronary reperfusion (IR). Larger, more active platelets were associated with intravascular plugging on an epicardial and myocardial level despite revascularisation of the infarct-related artery [3]. Larger platelets were also associated with larger myocardial infarct size and greater presence of microvascular obstruction, as well as poor pre- and post-interventional flow on epicardial and myocardial levels [5–7]. In a recently published study it was shown that increased platelet distribution width (PDW) was independently associated with CIN occurrence in patients with ACS [8].

We hypothesised that the pathophysiology of CI-AKI and IR, observed after invasive treatment of AMI, could be similar, related to platelets, and reflected in platelet count (PC) and platelet volume indices (PVI). Therefore, the aim of the presented work was to assess the relation between PC, PVI, IR, and CI-AKI in AMI-patients treated invasively. To date, there has not been any data published on this subject.

METHODS

Data acquisition

The clinical data from all AMI-patients treated with PCI were collected and prospectively recorded in a computerised database as part of a single-centre AMI registry. The recorded data included demographics, laboratory values, presence of concomitant diseases, characteristics of AMI, clinical assessment of diabetes, angiographic findings, and revascularisation procedure.

Selection of patients

The total study population consisted of 607 consecutive patients referred to our department because of AMI and treated invasively between January 2004 and December 2005. Clinical AMI criteria evaluated on admission were: chest pain persisting > 20 min, ST segment elevation of at least 0.1 mV in two or more contiguous electrocardiographic leads, or non-diagnostic electrocardiogram with enzymatic confirmation of AMI.

Comparative analyses were performed between patients with CI-AKI and without CI-AKI for the total study population (CI-AKI, n = 156; 25.7% vs. nCI-AKI, n = 451; 74.3%), for patients with diabetes mellitus (CI-AKI-DM, n = 56; 9.2% vs. nCI-AKI-DM, n = 123; 20.3%), and for patients with baseline kidney dysfunction (CI-AKI-BKD, n = 31; 5.1% vs. nCI-AKI-BKD, n = 67; 11.0%). Subjects with IR, who developed CI-AKI, were compared to the remaining patients (CI-AKI-IR, n = 47; 7.7% vs. controls, n = 560; 92.3%).

CI-AKI was defined as a rise in serum creatinine of at least 26.5 μmol/L (0.3 mg/dL) within 48 h after contrast exposure, or at least a 50% increase from the baseline value during index hospital stay [9, 10]. IR was defined as the lack of Thrombolysis in Myocardial Infarction (TIMI) flow grade 3 and/or lack of TIMI myocardial perfusion grade (MPG) 3 — the lack of optimal coronary and microvasculature flow despite no flow-limiting residual stenosis after PCI [2–4].

Baseline kidney dysfunction (BKD) was defined as estimated glomerular filtration rate (eGRF) < 60 mL/min/1.73 m^2. The eGFR was calculated using the serum creatinine value on admission before catheterisation, according to the abbreviated Modification of Diet in Renal Disease Study Group Equation proposed by the National Kidney Foundation [11]. Diabetes mellitus (DM) was diagnosed based on European Society of Cardiology and European Association for the Study of Diabetes criteria [12].

Ethics

All clinical data were obtained as a result of the diagnostic procedures and therapy, which were in accordance with the guidelines for myocardial infarction. All patients provided informed, written consent for hospitalisation, invasive treatment, and use of their data for research purposes. The study protocol was in line with ethical standards and was approved by the Institutional Review Board.

Catheterisation protocol and treatment

An invasive cardiologist and two technicians were on in-hospital duty 24 h a day. All patients before coronary angiography received a single dose of oral aspirin (300 mg), 300–600 mg of clopidogrel just before PCI, and 100 U/kg of intravenous heparin (additional boluses were given as appropriate to achieve activated clotting time > 250 ms). In all patients, coronary angiography and PCI of infarct-related artery were performed immediately after admission using standard techniques. The goal of PCI was to restore TIMI flow grade 3 with residual stenosis lower than 30%, which denotes a successful procedure. After PCI of infarct-related artery, TIMI flow grade and TIMI MPG were evaluated by the operator based on his visual assessment [13]. After the intervention, all patients received 75–150 mg of aspirin daily indefinitely, 75 mg of clopidogrel daily, as well as beta-blockers, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, and statins, if these agents had not been contraindicated.

Laboratory methods

Venous peripheral blood samples for the PC and PVI measurement were drawn on admission and analysed on an automated cell counter. Blood samples were taken into standardised tubes containing dipotassium ethylenedinitro tetraacetic acid

Table 1. Comparative analysis of demographics, and clinical and laboratory data between patients developing CI-AKI and patients without CI-AKI, in all subjects with acute myocardial infarction (n = 607)

	CI-AKI (n = 156)	nCI-AKI (n = 451)	P
Age [years]	64.1 ± 10.5	60.8 ± 11.1	0.001
Male	111 (71.2%)	325 (72.1%)	0.829
Hypertension	92 (59.0%)	223 (49.4%)	0.039
Hyperlipidaemia	95 (60.9%)	292 (64.7%)	0.400
Diabetes mellitus	56 (35.9%)	123 (27.3%)	0.043
Creatinine on admission [μmol/L]	95.1 ± 75.0	85.1 ± 26.1	0.016
Baseline kidney dysfunction	31 (19.9%)	67 (14.9%)	0.144
Uric acid [μmol/L]	387.8 ± 143.9	331.3 ± 98.2	< 0.001
Cardiogenic shock on admission	22 (14.1%)	20 (4.4%)	< 0.001
Ejection fraction < 35%	31 (19.9%)	46 (10.2%)	0.002
Multivessel coronary artery disease	104 (66.7)	274 (60.8)	0.190
TIMI < 3 and/or MPG < 3 after PCI of IRA	54 (34.6)	131 (29.0)	0.191
Contrast media volume [mL]	224.8 ± 87.9	219.6 ± 69.7	0.454
Platelet count [$\times 10^9$/L]	218.0 ± 64.8	207.6 ± 65.6	0.107
Mean platelet volume [fL]	11.4 ± 1.1	11.3 ± 1.0	0.442
Platelet volume distribution width [fL]	13.8 ± 2.3	13.7 ± 2.1	0.616
Platelet large cell ratio [%]	36.2 ± 8.9	35.5 ± 8.2	0.449

Values presented as mean ± standard deviation or percentage of subjects; CI-AKI — contrast induced acute kidney injury; IRA — infarct-related artery; MPG — myocardial perfusion grade; nCI-AKI — no contrast induced acute kidney injury; PCI — percutaneous coronary intervention; TIMI — Thrombolysis In Myocardial Infarction

(EDTA) and stored at room temperature. All measurements were performed within 30 minutes of blood collection.

Statistical analysis

Continuous parameters were expressed as means with standard deviations unless otherwise specified, and categorical variables were presented as numbers and percentages. Comparative analysis between groups was performed using the t-Student test for continuous variables and the χ^2 or Fisher's exact test, as appropriate, for dichotomous parameters. For total population as well as studied subgroups, multivariate logistic regression analyses were performed to reveal independent factors associated with CI-AKI. The results of the models were reported as odds ratios (OR) and 95% confidence intervals (95% CI). All tests were double-sided. P values < 0.05 were considered statistically significant. All analyses were performed using the software package Statistica (version 6.1, StatSoft Inc., Tulsa, OK, USA).

RESULTS

In the total study population patients with CI-AKI, when compared to subjects without CI-AKI, were significantly older, more often presented with hypertension, DM, ejection fraction < 35%, cardiogenic shock (CS) on admission, had higher uric acid and serum creatinine concentrations on admission. There were no differences between compared groups with respect to admission PC and PVI. The incidence of IR was similar in both groups. Detailed baseline characteristics for the total study population are shown in Table 1.

Within the DM group, CI-AKI-patients were significantly older, showed higher serum uric acid and creatinine concentrations on admission, more often presented with CS on admission, TIMI flow < 3, and/or MPG < 3 after PCI of infarct-related artery when compared to patients without CI-AKI. Among diabetics, in CI-AKI subjects, IR occurrence was almost two-fold higher than in nCI-AKI patients. Moreover, CI-AKI subjects showed higher PC. No differences in PVI were observed between the compared groups. Detailed baseline characteristics for subjects with DM are shown in Table 2.

Among subjects with BKD, CI-AKI-patients more often presented with CS on admission, ejection fraction < 35%, DM, and showed higher serum uric acid and creatinine concentrations on admission, when compared to patients without CI-AKI. Moreover, CI-AKI subjects showed higher PC. No differences in PVI were observed between the compared groups. The incidence of IR after the intervention was similar in both groups. Detailed baseline characteristics for subjects with BKD are shown in Table 3.

The comparative analysis between subjects who developed IR and CI-AKI and controls did not reveal any significant differences in relation to admission PC and PVI (Table 4).

Independent risk factors associated with CI-AKI within the total study population were: CS on admission (OR 3.94; CI 1.66–9.38; p = 0.002), ejection fraction at discharge

Table 2. Comparative analysis of demographic, clinical, and laboratory data between patients developing CI-AKI and patients without CI-AKI among subjects with diabetes mellitus (n = 179)

	CI-AKI (n = 56)	nCI-AKI (n = 123)	P
Age [years]	69.1 ± 7.8	66.0 ± 8.9	0.027
Male	37 (66.1%)	70 (56.9%)	0.245
Hypertension	40 (71.4%)	86 (69.9%)	0.839
Hyperlipidaemia	30 (53.6%)	79 (64.2%)	0.180
Creatinine on admission [μmol/L]	106.0 ± 76.6	90.0 ± 34.9	0.048
Baseline kidney dysfunction	21 (37.5%)	17 (24.4%)	0.074
Uric acid [μmol/L]	443.3 ± 176.8	368.7 ± 120.4	0.002
Cardiogenic shock on admission	14 (25.0%)	4 (3.3%)	< 0.001
Ejection fraction < 35%	11 (19.6%)	16 (13.0%)	0.254
Multivessel coronary artery disease	43 (76.8%)	86 (69.9%)	0.341
TIMI < 3 and/or MPG < 3 after PCI of IRA	27 (48.2%)	34 (27.6%)	0.008
Contrast media volume [mL]	228.7 ± 93.5	223.2 ± 86.4	0.701
Platelet count [× 10^9/L]	224.8 ± 62.8	197.9 ± 63.3	0.014
Mean platelet volume [fL]	11.4 ± 1.2	11.5 ± 1.0	0.661
Platelet volume distribution width [fL]	13.8 ± 2.4	14.2 ± 2.3	0.307
Platelet large cell ratio [%]	36.6 ± 9.4	37.0 ± 7.8	0.730

Abbreviations as in Table 1

Table 3. Comparative analysis of demographic, clinical and laboratory data between patients developing CI-AKI and patients without CI-AKI among subjects with baseline kidney dysfunction (n = 98)

	CI-AKI (n = 31)	nCI-AKI (n = 67)	P
Age [years]	68.9 ± 10.8	67.7 ± 8.5	0.559
Male	20 (64.5%)	34 (50.7%)	0.202
Hypertension	23 (74.2%)	39 (58.2%)	0.123
Hyperlipidaemia	16 (50.0%)	39 (58.2%)	0.449
Diabetes mellitus	21 (67.7%)	30 (44.8%)	0.037
Creatinine on admission [μmol/L]	177.5±138.4	125.4±37.7	0.005
Uric acid [μmol/L]	515.9 ± 189.3	411.0 ± 124.0	0.003
Cardiogenic shock on admission	11 (35.5%)	10 (14.9%)	0.023
Ejection fraction < 35%	8 (25.8%)	7 (10.4%)	0.052
Multivessel coronary artery disease	25 (80.6%)	45 (67.2%)	0.175
TIMI < 3 and/or MPG < 3 after PCI of IRA	14 (45.2%)	23 (34.3%)	0.309
Contrast media volume [mL]	217.9 ± 82.3	211.6 ± 79.2	0.718
Platelet count [× 10^9/L]	248.9 ± 86.5	202.5 ± 59.3	0.004
Mean platelet volume [fL]	11.3 ± 0.9	11.4 ± 1.1	0.663
Platelet volume distribution width [fL]	13.6 ± 1.6	13.9 ± 2.2	0.528
Platelet large cell ratio [%]	36.2 ± 7.3	36.7 ± 8.3	0.810

Abbreviations as in Table 1

(OR 0.96 per 1% increase; CI 0.94–0.98; p = 0.002), PC on admission was not independently associated with CI-AKI, only trend towards higher risk of CI-AKI was observed. Independent factors associated with CI-AKI in patients with DM were: age (OR 1.05; CI 1.0–1.09; p = 0.04), CS on admission (OR 10.0; CI 2.60–38.5; p < 0.001), and PC on admission (per one unit increase OR 1.006; CI 1.0–1.01; p = 0.04). Independent factors associated with CI-AKI in patients with BKD were: CS on admission (OR 6.36; CI 1.2–33.75; p = 0.03) and PC on admission (per one unit increase OR 1.01; CI 1.0–1.02; p = 0.02).

Similar analyses were performed to identify independent predictors of no-reflow; however, no platelet-related risk factors for no-reflow were found in the study population or in the analysed subgroups.

Table 4. Comparative analysis of platelet parameters in patients developing contrast-induced acute kidney injury and impaired coronary reperfusion (CI-AKI-IR) with control group

	CI-AKI-IR (n = 47)	Controls (n = 560)	P
Platelet count [× 10^9/L]	217.4 ± 65.8	209.5 ± 65.5	0.440
Mean platelet volume [fL]	11.2 ± 1.2	11.3 ± 1.0	0.734
Platelet volume distribution width [fL]	13.6 ± 2.5	13.7 ± 2.2	0.675
Platelet large cell ratio [%]	35.2 ± 9.6	35.7 ± 8.2	0.687

Values presented as mean ± standard deviation.

DISCUSSION

This work demonstrated that in the studied groups of AMI patients, contrast-induced nephropathy occurrence was not associated with admission PVI. Higher PC was observed in CI-AKI subjects with diabetes or BKD. Moreover, an increase in admission PC was an independent risk factor for CI-AKI development. This association had not been found in prior studies.

Published studies showed that larger, more active platelets play a role in the "no reflow" phenomenon. We hypothesised that platelets with more prothrombotic potential, responsible for coronary vascular plugging observed in no reflow, could also cause CI-AKI in a similar manner. However, in patients with CI-AKI and IR after invasive treatment, no associations with platelet parameters were found. Therefore, any similarities in the pathogenesis of these two diseases are not reflected in PC or in PVI.

In the presented study, patients who developed contrast-induced nephropathy had more often well-established CI-AKI risk factors than nCI-AKI controls — CS, diabetes, worse baseline renal function, and older age [10, 13]. CS was the most important independent risk factor for CI-AKI in the total study population and analysed subgroups. Higher PC in subjects with DM or baseline kidney dysfunction, who developed CI-AKI, had been also observed in patients with longer diabetes duration complicated with diabetic nephropathy [14] or insulin resistance [15], in women with metabolic syndrome [16]. The obtained data indicated that the coexistence of renal dysfunction and diabetes was associated with increased PC, but sex differences were not confirmed. In a recently published study by Sahin et al. [8] it was shown that an increase in PDW was independently associated with CI-AKI occurrence, an observation not found in the presented study. The investigated population in Sahin's study consisted of patients with more favourable clinical presentation of ACS, presumably without patients with CS. Moreover, the incidence of AMI as well as frequency of invasive treatment was not shown. Therefore, different results relating to PDW could be caused by different platelet activation in different presentations of ACS. Platelet count was not analysed in Sahin's study [8].

Limitations of the study

This study was designed as a prospective single-centre observational analysis, which could potentially have biased the results. The study groups were relatively small.

CONCLUSIONS

Considering the results, the authors concluded that the pathophysiology of CI-AKI seems to be different to that of IR. Any similarities in the pathophysiology of CI-AKI and IR were not reflected in platelet parameters. CI-AKI development was not related to PVI; however, higher PC was an independent risk factor for CI-AKI in patients with BKD or diabetes.

Conflict of interest: *Radosław Lenarczyk — consultant fees: Medtronic, Biotronik, St. Jude Medical, speaker fees: Boehringer Ingelheim; Beata Średniawa — consultant fees: Medtronic Bakken Research Center, Bristol Myers Squibb, Pfizer, speaker fees: Boehringer Ingelheim, Servier, MSD, Adamed, Berlin Chemie, Sandoz, Reynolds Medical; Zbigniew Kalarus — speaker fees: Pfizer, Novonordisk, Eli Lilly, Boehringer Ingelheim, scientific conferences and congresses sponsorship: St. Jude Medical, Servier, Medtronic, consultant fees: Boehringer Ingelheim.*

References

1. Tumlin J, Stacul F, Adam A et al. CIN Consensus Working Panel. Pathophysiology of contrast-induced nephropathy. Am J Cardiol, 2006; 98: 14K–20K.
2. Kang S, Yang Y. Coronary microvascular reperfusion injury and no-reflow in acute myocardial infarction. Clin Invest Med, 2007; 30: E133–E145.
3. Huczek Z, Kochman J, Filipiak KJ et al. Mean platelet volume on admission predicts impaired reperfusion and long-term mortality in acute myocardial infarction treated with primary percutaneous coronary intervention. J Am Coll Cardiol, 2005; 46: 284–290.
4. Ndrepepa G, Tiroch K, Fusaro M et al. 5-year prognostic value of no-reflow phenomenon after percutaneous coronary intervention in patients with acute myocardial infarction. J Am Coll Cardiol, 2010; 55: 2383–2389. doi: 10.1016/j.jacc.2009.12.054.
5. Fabregat-Andrés Ó, Cubillos A, Ferrando-Beltrán M et al. Mean platelet volume is associated with infarct size and microvascular obstruction estimated by cardiac magnetic resonance in ST segment elevation myocardial infarction. Blood Coagul Fibrinolysis, 2013; 24: 424–427. doi: 10.1097/MBC.0b013e32835d9bca.
6. Sarli B, Baktir AO, Saglam H et al. Mean platelet volume is associated with poor postinterventional myocardial blush grade in patients with ST-segment elevation myocardial infarction.

Coron Artery Dis, 2013; 24: 285–289. doi: 10.1097/MCA..0b013e-328360eff6.
7. Elbasan Z, Gür M, Sahin DY et al. Association of mean platelet volume and pre- and postinterventional flow with infarct-related artery in ST-segment elevation myocardial infarction. Angiology, 2013; 64: 440–446. doi: 10.1177/0003319712455685.
8. Sahin I, Karabulut A, Avci II et al. Contribution of platelets indices in the development of contrast-induced nephropathy. Blood Coagul Fibrinolysis, 2015; 26: 246–249. doi: 10.1097/MBC.0000000000000107.
9. Kellum JA, Lameire N; for the KDIGO AKI Guideline Work Group. Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (Part 1). Crit Care, 2013; 17: 204. doi: 10.1186/cc11454.
10. Lameire N, Kellum JA; for the KDIGO AKI Guideline Work Group. Contrast-induced acute kidney injury and renal support for acute kidney injury: a KDIGO summary (Part 2). Crit Care, 2013; 17: 205. doi: 10.1186/cc11455.
11. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis, 2002; 39: S1–S266.
12. ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed In collaboration with the EASD: the Task Force on diabetes, pre-diabetes, and cardiovascular diseases of the European Society of Cardiology (ESC) and developer in collaboration with the European Association for the Study of Diabetes (EASD). Eur Heart J, 2013; 34: 3035–3087. doi: 10.1093/eurheartj/eht108.
13. TIMI Study Group. TIMI Definitions for Commonly Used Terms in Clinical Trials. http://www.timi.org.
14. Sterner G, Carlson J, Ekberg G. Raised platelet levels in diabetes mellitus complicated with nephropathy. J Intern Med, 1998; 244: 437–441.
15. Taniguchi A, Fukushima M, Seino Y et al. Platelet count is independently associated with insulin resistance in non-obese Japanese type 2 diabetic patients. Metabolism, 2003; 52: 1246–1249.
16. Park BJ, Shim JY, Lee HR et al. The relationship of platelet count, mean platelet volume with metabolic syndrome according to the criteria of the American Association of Clinical Endocrinologists: a focus on gender differences. Platelets, 2012; 23: 45–50. doi: 10.3109/09537104.2011.589014.

Liczba i wskaźniki objętości płytek krwi u chorych z wywołanym kontrastem ostrym uszkodzeniem nerek i zawałem serca leczonych inwazyjnie

Paweł Francuz[1], Jacek Kowalczyk[1], Ryszard Swoboda[2], Katarzyna Przybylska-Siedlecka[1], Monika Kozieł[1], Tomasz Podolecki[1], Andrzej Świątkowski[1], Radosław Lenarczyk[1], Beata Średniawa[1], Zbigniew Kalarus[1]

[1]Katedra Kardiologii, Wrodzonych Wad Serca i Elektroterapii, Oddział Kliniczny Kardiologii, Śląskie Centrum Chorób Serca, Śląski Uniwersytet Medyczny, Zabrze

[2]Śląski Uniwersytet Medyczny, Katowice

Streszczenie

Wstęp: Etiologia ostrego, wywołanego kontrastem uszkodzenia nerek (CI-AKI) nie jest w pełni poznana. Autorzy postawili hipotezę, że patofizjologia CI-AKI i upośledzonej reperfuzji wieńcowej (IR) po inwazyjnym leczeniu ostrego zawału serca (AMI) może być podobna, zależna od liczby płytek krwi (PC) i parametrów objętości płytek (PVI).

Cel: Celem pracy była ocena zależności między PC, PVI, IR a rozwojem CI-AKI u pacjentów z AMI leczonych inwazyjnie.

Metody: Jednoośrodkowym badaniem objęto 607 kolejno przyjmowanych do Kliniki chorych z AMI leczonych inwazyjnie. Analizy porównawcze przeprowadzono między pacjentami z CI-AKI i bez tej nefropatii (nCI-AKI) w obrębie całej populacji (CI-AKI, n = 156; 25,7% *vs*. nCI-AKI, n = 451; 74,3%), dla chorych na cukrzycę (CI-AKI-DM, n = 56; 9,2% *vs*. nCI-AKI-DM, n = 123; 20,3%), dla osób z upośledzoną funkcją nerek przy przyjęciu (CI-AKI-BKD, n = 31; 5,1% *vs*. nCI-AKI-BKD, n = 67; 11,0%). Pacjentów z IR, u których rozwinęło się CI-AKI, porównano z pozostałymi chorymi (CI-AKI-IR, n = 47; 7,7% *vs*. grupa kontrolna, n = 560; 92,3%). Dla całej populacji badanej, jak również badanych podgrup wykonano modele regresji logistycznej w celu wyłonienia niezależnych czynników ryzyka rozwoju CI-AKI. Wyniki analiz wieloczynnikowych przedstawiono jako ilorazy szans (OR) i 95-procentowe przedziały ufności (95% CI).

Wyniki: Liczba płytek krwi była wyższa u grupie CI-AKI-DM (224,8 ± 62,8 × 10^9/l *vs*. 197,9 ± 63,3 × 10^9/l; p = 0,014) i CI-AKI-BKD (248,9 ± 86,5 × 10^9/l *vs*. 202,5 ± 59,3 × 10^9/l; p = 0,004) niż w odpowiednich grupach kontrolnych. W obrębie badanych grup nie było różnic między pacjentami z CI-AKI i nCI-AKI w odniesieniu do PVI. Porównując grupę CI-AKI-IR z grupą kontrolną, nie stwierdzono różnic w PC czy PVI. IR częściej obserwowano w grupie CI-AKI niż w grupie bez tej nefropatii tylko u pacjentów z cukrzycą (48,2% *vs*. 27,6%; p = 0,008). Większa liczba płytek krwi przy przyjęciu do szpitala była niezależnie związana ze zwiększonym ryzykiem rozwoju CI-AKI w grupie pacjentów z cukrzycą (wzrost o 1 jednostkę wiązał się z OR 1,006; CI 1,0–1,01; p = 0,04) lub upośledzoną funkcją nerek przy przyjęciu (wzrost o 1 jednostkę wiązał się z OR 1,01; CI 1,0–1,02; p = 0,02).

Wnioski: Potencjalne podobieństwa w patofizjologii CI-AKI i IR nie zostały odzwierciedlone w PVI. Rozwój CI-AKI nie był związany z PVI, jednak wyższa wartość PC była niezależnym czynnikiem ryzyka CI-AKI u pacjentów z cukrzycą lub nieprawidłową funkcją nerek przy przyjęciu.

Słowa kluczowe: ostry zawał serca, wywołane kontrastem ostre uszkodzenie nerek, cukrzyca, liczba płytek krwi, wskaźniki objętości płytek

Kardiol Pol 2015; 73, 7: 520–526

Adres do korespondencji:
lek. Paweł Francuz, Katedra Kardiologii, Wrodzonych Wad Serca i Elektroterapii, Oddział Kliniczny Kardiologii, Śląskie Centrum Chorób Serca, Śląski Uniwersytet Medyczny, ul. Curie-Sklodowskiej 9, 41–800 Zabrze, e-mail: pawel.francuz@wp.pl
Praca wpłynęła: 12.09.2014 r. **Zaakceptowana do druku:** 22.01.2015 r. **Data publikacji AoP:** 06.03.2015 r.