Original paper

Risk factors of acute kidney injury during hospitalization in acute ischaemic stroke patients undergoing mechanical thrombectomy

Paweł Wrona[1], Katarzyna Sawczyńska[1], Dominik Wróbel[2], Kaja Zdrojewska[2], Mateusz Giełczyński[2], Paweł Mizera[2], Paweł Brzegowy[3], Tadeusz Popiela[3], Agnieszka Słowik[1], Marcin Krzanowski[4]

[1]Department of Neurology, Jagiellonian University Medical College, Krakow, Poland
[2]Student Scientific Group in Cerebrovascular Diseases, Jagiellonian University Medical College, Krakow, Poland
[3]Department of Radiology, Jagiellonian University Medical College, Krakow, Poland
[4]Department of Nephrology and Dialysis, Jagiellonian University Medical College, Krakow, Poland

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A b s t r a c t

Introduction: Acute kidney injury (AKI) seems to worsen the prognosis of acute ischaemic stroke (AIS) patients treated with mechanical thrombectomy (MT). At the same time, the procedure of MT increases AKI risk by iodinated contrast use. Identification of factors predisposing to AKI after MT is important for recognizing vulnerable patients and successful prevention.

Aim: To identify factors associated with the occurrence of AKI during hospitalization in MT-treated AIS patients.

Material and methods: The study included all AIS patients treated with MT in the University Hospital in Krakow from 2019 to 2021. The diagnosis of AKI during hospitalisation was based on serum creatinine concentration levels, according to the Kidney Disease Improving Global Outcomes guidelines. We compared patients with and without AKI in terms of age, sex, comorbidities, stroke course and laboratory test results at admission. We identified factors associated with the occurrence of AKI using univariate logistic regression analysis, with significant variables subsequently added to the multivariate analyses.

Results: Among 593 MT-treated AIS patients the incidence of AKI during hospitalisation was 12.6%. AKI development was associated with diabetes, chronic kidney disease, total volume of iodinated contrast obtained during hospitalisation, posterior circulation stroke, lack of intravenous thrombolysis, and laboratory test results at admission: haemoglobin, glucose, urea, potassium, and creatinine. Total contrast volume and urea level were the most important independent risk factors associated with occurrence of AKI.

Conclusions: AKI is common in MT-treated AIS patients. There is a need to establish a protocol for decreasing the risk of AKI in AIS patients undergoing MT and, in case it occurs, a procedure for its treatment.

Key words: acute kidney injury, ischaemic stroke, mechanical thrombectomy, endovascular stroke treatment.

S u m m a r y

Acute kidney injury (AKI) may be associated with worse prognosis in patients with acute ischaemic stroke treated with mechanical thrombectomy (MT); therefore identification of risk factors of AKI is important for recognizing vulnerable patients and successful prevention. Within the group of 593 AIS patients treated with MT in the University Hospital in Krakow, Poland, AKI during hospitalization occurred in 12.6%. Factors associated with the development of AKI identified by univariate logistic regression analysis were: diabetes mellitus, chronic kidney disease, total iodinated contrast volume received during hospital stay, posterior circulation stroke, lack of treatment with intravenous thrombolysis, as well as haemoglobin, glucose, urea, potassium, and creatinine levels at admission, with total contrast volume and urea level being the most important independent risk factors found in multivariate analyses.

Corresponding author:
Paweł Wrona, Department of Neurology, Jagiellonian University Medical College, Krakow, Poland, e-mail: pawel.wrona@uj.edu.pl
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Introduction

Mechanical thrombectomy (MT) is an established treatment method of acute ischaemic stroke (AIS) caused by a large vessel occlusion [1]. Although highly efficient, it is inevitably associated with risk factors of acute kidney injury (AKI), such as iodine contrast administration for computed tomography (CT) procedures needed for selection for MT and for MT itself, as it is a catheter-based procedure [2]. AKI is a relatively common complication observed in MT-treated AIS patients, estimated in a recent systematic review with meta-analysis to occur in approximately 7% of cases [3], with the percentage of patients needing renal replacement therapy being much lower (0.2% in another meta-analysis) [4]. Even though some studies suggest that MT is a risk factor for AKI in stroke patients [5], experts point out that, given the very low risk of end-stage kidney failure, MT should not be delayed or avoided due to the fear of kidney damage in patients otherwise eligible for the procedure [4, 6].

Data about the impact of AKI on patient outcomes is scarce, but so far evidence suggests that it is associated with worse short- and long-term prognosis [7–12]. Therefore, identification of the risk factors of AKI after MT is important for identifying vulnerable patients and successful prevention. At the same time, not many studies on this subject have been published and their results are not unanimous, making it important to perform further analyses on this topic.

Aim

The aim of the study was to determine the frequency of AKI in AIS patients treated by MT, and to identify factors associated with the occurrence of AKI during hospitalization in this group of patients.

Material and methods

We performed a retrospective analysis of prospectively collected data of AIS patients treated with MT in the Comprehensive Stroke Centre of the University Hospital in Krakow, Poland, from 2019 to 2021.

The following parameters were analysed: age, sex and profile of risk factors known at admission (arterial hypertension, atrial fibrillation, diabetes mellitus, history of stroke or transient ischaemic attack, hypercholesterolaemia, history of myocardial infarction, obesity, history of smoking, alcohol abuse, carotid artery atherosclerosis, chronic kidney disease, multiple myeloma, and liver cirrhosis). We also analysed the following treatments with known nephrotoxic potential used before stroke: angiotensin-conversing enzyme inhibitors (ACEI), angiotensin receptor blockers (ARB), nonsteroidal anti-inflammatory drugs (NSAID), antiplatelets, spironolactone, eplerenone and metformin. We estimated the amount of iodinated contrast received during hospitalization based on mean contrast volumes administered in our centre during different procedures. We noted the first serum laboratory test results after admission to the stroke ward: haemoglobin, platelet count, glucose, total cholesterol, urea, creatinine, sodium and potassium levels. We counted mean arterial blood pressure (MAP) at admission.

We also gathered information concerning AIS course: ischaemic lesion location (anterior or posterior circulation), neurological deficit at admission assessed using the National Institutes of Health Stroke Scale (NIHSS) as well as infarct and penumbra volumes estimated using postprocessing analysis of perfusion computed tomography (perfusion-CT) with the iRAPID program [13]. Treatment with IVT, time from stroke onset to groin puncture and radiological effect of mechanical thrombectomy assessed with the modified Treatment in Cerebral Ischaemia (mTICI) scale were also analysed. Full reperfusion (good radiological effect of MT) was defined as an mTICI score of 2b-3. The occurrence of intracerebral haemorrhage secondary to reperfusion therapy (sICH) was noted.

We identified patients who developed AKI during hospitalisation based on KDIGO guidelines [14], with AKI defined as a rise in serum creatinine concentration of ≥ 26.5 µmol/l within 48 h or ≥ 1.5-fold increase within 7 days.

Jagiellonian University Bioethics Committee approval was given for the study (decision number 1072.6120.118.2020). The study was financially supported by the iBioStroke grant (Identification and clinical validation of biomarkers for long-term outcome after cerebral ischaemia, ERA-NET-NEURON/21/2020, K/NCB/00057).

Statistical analysis

We compared data between groups of patients with and without AKI. Statistical analysis was performed using the PS Imago Pro 9.0 program. Comparisons of categorical data were made using the χ^2 test and those of continuous data using the Mann-Whitney *U*-test (as assessment of normality with the Kolmogorov-Smirnov test revealed non-normal data distribution). Differences were considered statistically significant when the *p*-value was less than 0.05. Variables differing significantly between the groups were then analysed using univariate logistic regression to confirm their association with development of AKI during hospitalization. Variables positively associated with the development of AKI were subsequently included in a multivariate analysis. As the sample size allowed us only to include seven variables in the multivariate analysis, two different models were presented – one including variables considered most relevant from a clinical point of view, and another one including variables with the lowest *p*-value identified during univariate analysis.

Results

The study included 593 MT-treated AIS patients, from which 75 (12.6%) fulfilled AKI criteria during hospitalisation.

Patients developing AKI during hospitalisation, compared to patients without AKI, more commonly suffered from diabetes mellitus (32.0% vs. 18.1%, $p = 0.006$) and chronic kidney disease (19.2% vs. 8.6%, $p = 0.007$). They received higher iodinated contrast volumes during hospitalisation: median of 200 (IQR = 80) vs. 200 (IQR = 40) ml, $p = 0.024$. The ischaemic lesion was more commonly located in the posterior circulation (21.3% vs. 12.5%, $p = 0.047$). AKI (+) patients were less frequently treated with IVT (45.3% vs. 58.7%, $p = 0.034$). There were also statistically significant differences in laboratory test results after admission, with AKI (+) patients initially presenting with lower haemoglobin (12.5 (IQR = 2.7) vs. 13.1 (IQR = 2.3) g/dl, $p = 0.038$), higher platelet count (221.5 (IQR = 71.3) vs. 212 (IQR = 86.3) × 10^3/µl, $p = 0.018$), higher glucose (7 (IQR = 3.7) vs. 5.9 (IQR = 2.4) mmol/l, $p = 0.002$), urea (7.5 (IQR = 5.1) vs. 5.5 (IQR = 2.8) mmol/l, $p < 0.001$), potassium (4.3 (IQR = 0.6) vs. 4.1 (IQR = 0.5) mmol/l, $p = 0.005$) and creatinine concentration (92.1 (IQR = 38.6) vs. 78 (IQR = 26.2) µmol/l, $p < 0.001$). Group comparison is presented in Table I.

The above-mentioned variables were assessed using univariate logistic regression analysis and, apart from the platelet count, all of them turned out to be associated with the development of AKI during hospitalization – detailed results are presented in Table II.

Sample size allowed us only to include seven variables in the multivariate analysis. In the first model, presented in Table III, we included variables that we considered to be most relevant from a clinical point of view: diabetes mellitus, chronic kidney disease, iodinated contrast volume received during hospitalisation, posterior circulation stroke, IVT, and first serum urea and creatinine concentration result after admission to the stroke ward. In this model, resulting with $R^2 = 0.165$, factors independently associated with the development of AKI were total volume of iodinated contrast received during hospitalization (OR = 1.007, 95% CI: 1.003–1.011, $p = 0.001$), treatment with IVT (OR = 0.551, 95% CI: 0.307–0.989, $p = 0.046$) and first serum urea level after admission (OR = 1.224, 95% CI: 1.104–1.356, $p < 0.001$).

In the second model, including seven variables with the lowest p-values identified during univariate analysis (as presented in Table IV) independent factors associated with the development of AKI during hospitalization were: total volume of iodinated contrast received during hospitalization (OR = 1.007, 95% CI: 1.00–1.012, $p = 0.002$) and first laboratory test results after admission: urea (OR = 1.223, 95% CI: 1.097–1.363, $p < 0.001$) and haemoglobin level (OR = 0.846, 95% CI: 0.726–0.985, $p = 0.031$). R^2 of this model was 0.177.

Discussion

In our study AKI occurred in 12.6% of MT-treated AIS patients, which was a higher frequency than presented in the results of a recent meta-analysis including twenty-two studies, that showed the pooled incidence of AKI after MT to be 7%. However, heterogeneity was high and in particular studies it ranged from 1% to even 33% [3].

Studies evaluating potential risk factors for the development of AKI after MT have so far also produced mixed results. In the works by Delgado Acosta *et al.* [6] and Diprose *et al.* [10] including 189 and 333 MT-treated patients respectively, contrast-induced nephropathy was independently associated with worse kidney function at admission. In a study by Hu *et al.*, including 184 patients, creatinine increase after MT was independently associated with diabetes [15]. In a study by Myung *et al.* independent risk factors for post-contrast AKI among 601 MT-treated AIS patients were older age, chronic kidney disease, serum albumin level and the use of NSAID, ACEI, ARB, β-blockers, statins, and insulin after MT [16]. Yoo *et al.*, in a study of 601 MT-treated patients, concluded that factors independently associated with AKI were diabetes, total contrast dose and unsuccessful reperfusion [12]. In a study by Yamamoto *et al.*, including 80 patients after MT, contrast-induced nephropathy was independently predicted by white blood cell count and the quotient of contrast volume and estimated glomerular filtration rate [17]. In a study by Weber *et al.* among 1017 MT-treated AIS patients those who developed AKI compared to those without AKI significantly more often suffered from diabetes and chronic kidney disease and more frequently had a tandem occlusion, but multivariate logistic regression analysis revealed no significant independent risk factors for AKI [7]. In a study by Loh *et al.,* including 99 patients, higher rates of diabetes mellitus, chronic renal insufficiency, preadmission statin and NSAID use were observed in patients who developed contrast-associated AKI, but those results were not statistically significant, although there was significantly higher serum creatinine concentration at admission in the AKI (+) group [18]. In a study by Laible *et al.*, including 1169 subjects, multivariate analysis surprisingly showed that developing post-contrast AKI within the first 48 h after MT was associated with higher glomerular filtration rate at admission and higher percentage of patients being treated with IVT [9]. A meta-analysis by Jhou *et al.* including 15 studies with over 27 000 participants showed that a possible risk factor of AKI in MT-treated patients was diabetes, with baseline renal function and the volume of contrast not being significantly associated with AKI [4].

The above-mentioned differences of results between studies may derive from use of different definitions of AKI as well as different time frames for in-hospital observation. In our study we decided to use KDIGO criteria, in which AKI is defined as an increase of serum creati-

Table I. Comparison of groups of patients with and without AKI during hospitalization

Parameter	AKI (+)	AKI (–)	*P*-value	*N*
Demographics:				
Age [years], median (IQR)	71 (IQR = 15)	70 (IQR = 17)	0.85	593
Female sex, *n* (%)	34 (45.3)	248 (47.9)	0.71	593
Comorbidities:				
Arterial hypertension, *n* (%)	51 (68.0)	348 (67.2)	0.90	593
Atrial fibrillation, *n* (%)	17 (22.7)	154 (29.7)	0.22	593
Diabetes mellitus, *n* (%)	24 (32.0)	94 (18.1)	0.006	593
History of stroke/TIA, *n* (%)	14 (18.7)	57 (11.0)	0.06	593
Hypercholesterolaemia, *n* (%)	14 (18.7)	80 (15.5)	0.50	592
History of myocardial infarction, *n* (%)	13 (17.3)	62 (12.0)	0.20	593
Obesity, *n* (%)	14 (18.7)	102 (19.7)	0.88	593
History of smoking, *n* (%)	14 (18.7)	110 (21.3)	0.65	592
Alcohol abuse, *n* (%)	9 (12.0)	32 (6.2)	0.08	593
Carotid artery atherosclerosis, *n* (%)	12 (16.0)	89 (17.2)	0.87	593
Chronic kidney disease, *n* (%)	14 (19.2)	44 (8.6)	0.007	587
Multiple myeloma, *n* (%)	0 (0.0)	0 (0.0)	–	588
Cirrhosis, *n* (%)	0 (0.0)	2 (0.4)	1.00	588
Medication:				
ACEI before hospitalisation, *n* (%)	22 (33.3)	131 (26.9)	0.31	553
ARB, *n* (%)	4 (6.1)	61 (12.5)	0.16	553
NSAIDs, *n* (%)	23 (34.3)	134 (27.4)	0.25	556
Antiplatelet, *n* (%)	24 (32.9)	123 (24.7)	0.15	570
Spironolactone, *n* (%)	7 (10.4)	28 (5.7)	0.17	556
Eplerenone, *n* (%)	2 (3.0)	15 (3.1)	1.00	556
Metformin, *n* (%)	3 (4.5)	42 (8.6)	0.34	555
Total contrast volume during hospitalisation [ml] median (IQR)	220 (IQR = 80)	220 (IQR = 40)	0.024	593
Disease course:				
Posterior circulation stroke, *n* (%)	16 (21.3)	65 (12.5)	0.047	593
NIHSS score at admission, median (IQR)	16 (IQR = 10)	16 (IQR = 9)	0.27	593
Penumbra volume [ml] median (IQR)	83 (IQR = 84)	90 (IQR = 75)	0.28	529
Infarct volume [ml] median (IQR)	10 (IQR = 35)	7 (IQR = 25)	0.31	521
IVT, *n* (%)	34 (45.3)	304 (58.7)	0.034	593
Time from onset to groin puncture [min] median (IQR)	300 (IQR = 140)	290.5 (IQR = 145)	0.83	593
Full reperfusion, *n* (%)	61 (81.3)	459 (88.8)	0.09	593
sICH, *n* (%)	21 (30.4)	105 (21.1)	0.09	593
Laboratory test results:				
Haemoglobin [g/dl] median (IQR)	12.5 (IQR = 2.7)	13.1 (IQR = 2.3)	0.038	581
Platelet count [× 10^3/µl] median (IQR)	221.5 (IQR = 71.3)	212 (IQR = 86.3)	0.018	580
Glucose [mmol/l] median (IQR)	7 (IQR = 3.7)	5.9 (IQR = 2.4)	0.002	547
Total cholesterol [mmol/l] median (IQR)	4.2 (IQR = 2.1)	4.1 (IQR = 1.4)	0.83	560
Urea [mmol/l] median (IQR)	7.5 (IQR = 5.1)	5.5 (IQR = 2.8)	< 0.001	564
Sodium [mmol/l] median (IQR)	139 (IQR = 4)	139 (IQR = 4)	0.39	565
Potassium [mmol/l] median (IQR)	4.3 (IQR = 0.6)	4.1 (IQR = 0.5)	0.005	593
Creatinine [µmol/l] median (IQR)	92.1 (IQR = 38.6)	78 (IQR = 26.2)	<0.001	523
MAP [mm Hg] median (IQR)	110 (IQR = 23)	106 (IQR = 20)	0.23	374

TIA – transient ischaemic attack, ACEI – angiotensin-conversing enzyme inhibitors, ARB – angiotensin receptor blockerS, NSAIDs – nonsteroidal anti-inflammatory drugs, NIHSS – National Institutes of Health Stroke Scale, IVT – intravenous thrombolysis, sICH – secondary intracerebral haemorrhage, MAP – mean arterial blood pressure.

nine concentration of at least 26 µmol/l within 48 h or at least 1.5 times baseline within 7 days, or as urine output of less than 0.5 ml/kg/h for 6 h [14]. As we could not retrospectively analyse urine output, we limited our analysis to the serum creatinine concentration. The most important limitation of our study is the fact that we only analysed laboratory tests available after admission to the stroke unit, as pre-admission results were obtained using different laboratory methods and were therefore not comparable. In some cases, the first creatinine concentration result was obtained only after treatment with MT.

Other limitation of our study was that we retrospectively analysed prospectively collected data that were initially focused on cognitive outcomes after stroke as a part of the study "Identification and clinical validation of biomarkers for long-term outcome after cerebral ischaemia (IBioStroke)" (https://www.neuron-eranet.eu/wp-content/uploads/iBioStroke.pdf), therefore making us unable to analyse various factors that may affect AKI, such as the level of dehydration at admission. What is more, the total amount of contrast received during hospitalisation was estimated based on average amounts used for different procedures, and might have been different in individual patients, which could have influenced the results.

In our study AKI was associated with total iodinated contrast dose as well as diabetes and chronic kidney disease (which may result from diabetes) – those associations have been reported in previous studies, as mentioned before. Significant differences in laboratory test results (higher creatinine concentration, urea, potassium and glucose levels and lower haemoglobin) are probably a direct effect of the latter two chronic diseases. We hypothesize that the lower percentage of AKI among patients treated with IVT may result from a smaller burden of comorbidities (possible contraindications for IVT) in this group. The reason for the association of AKI with posterior circulation stroke is unclear – it may be hypothesized that it is due to severe multiorgan complications observed in some patients with brainstem strokes, especially that posterior circulation stroke has not been proven to be an independent factor associated with AKI, but we have no evidence to prove that theory.

A few studies evaluating the impact of AKI on the prognosis of MT-treated AIS patients showed that AKI is associated with higher in-hospital mortality [7–9] and worse functional outcome at discharge[8] as well as in a 3-month follow-up [9–12]. Therefore, identification of patients at risk of AKI, careful monitoring of their kidney function and immediate treatment of kidney injury are most likely beneficial for MT treatment prognosis.

Conclusions

AKI is a relatively common complication seen in MT-treated AIS patients. Its prevention, early detection and successful treatment may improve the patients' outcomes. Based on the results of our study we recommend paying close attention to kidney function in patients with high urea levels at admission (due to CKD or dehydration). The procedure of MT, reversing the consequences of stroke in a significant number of AIS patients, requires administration of iodine contrast without delay, irrespectively of the future risk of AKI. Since AKI is a common complication (12.6% of stroke patients treated by MT), and the infusion of iodine contrast is an identified risk factor, it is necessary to establish a protocol for decreas-

Table II. Univariate logistic regression analysis showing factors associated with development of AKI during hospitalization

Parameter	*P*-value	OR	95% CI
Diabetes mellitus	0.006	2.123	1.244–3.621
Chronic kidney disease	0.006	2.535	1.311–4.902
Total contrast volume [ml]	0.006	1.005	1.001–1.009
Posterior circulation stroke	0.041	1.890	1.026–3.480
Intravenous thrombolysis	0.030	0.584	0.359–0.950
Haemoglobin [g/dl]	0.009	0.851	0.753–0.961
Platelet count [× 10^3/µl]	0.099	1.002	1.000–1.005
Glucose [mmol/l]	0.047	1.058	1.001–1.119
Urea [mmol/l]	< 0.001	1.231	1.149–1.319
Potassium [mmol/l]	0.003	2.052	1.277–3.279
Creatinine [µmol/l]	0.002	1.008	1.003–1.014

Table III. Multivariate analysis of factors most relevant for the development of AKI from a clinical point of view

Parameter	*P*-value	OR	95% CI
Diabetes mellitus	0.104	1.678	0.899–3.135
Chronic kidney disease	0.607	0.779	0.301–2.018
Total contrast volume [ml]	0.001	1.007	1.003–1.011
Posterior circulation stroke	0.249	1.583	0.726–3.453
Intravenous thrombolysis	0.046	0.551	0.307–0.989
Urea [mmol/l]	< 0.001	1.224	1.104–1.356
Creatinine [µmol/l]	0.794	1.001	0.995–1.007

Table IV. Multivariate analysis including seven variables with the lowest *p*-values identified during univariate analysis

Parameter	*P*-value	OR	95% CI
Diabetes	0.067	1.831	0.958–3.498
Chronic kidney disease	0.420	0.665	0.246–1.795
Total contrast volume [ml]	0.002	1.007	1.003–1.012
Urea [mmol/l]	0.001	1.223	1.097–1.363
Potassium [mmol/l]	0.353	1.341	0.722–2.490
Creatinine [µmol/l]	0.967	1.000	0.994–1.006
Haemoglobin [g/dl]	0.031	0.846	0.726–0.985

ing the risk of AKI in AIS patients treated with MT and, in case it appears, a procedure for its treatment.

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Conflict of interest

The authors declare no conflict of interest.

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