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Acute kidney injury negatively affects short and long-term outcomes of mechanical thrombectomy in acute ischaemic stroke

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Abstract

Aim of study. We aimed to assess the impact of acute kidney injury (AKI) during hospitalisation on short- and long-term outcomes of mechanical thrombectomy (MT) in patients with acute ischaemic stroke (AIS).

Clinical rationale for study. AKI is a common complication in AIS patients treated with MT. Some studies examining its impact on prognosis have shown an association of AKI with worse MT outcomes, but observations exceeding three months are lacking.

Material and methods. To this observational cohort study, we included all AIS patients treated with MT in the University Hospital in Krakow from 2019 to 2021. AKI during hospitalisation was diagnosed based on serum creatinine concentration levels according to the KDIGO (Kidney Disease Improving Global Outcomes) guidelines. We compared patients with and without AKI in terms of mortality and functional outcome (assessed with modified Rankin scale, mRS) at discharge, and at 90 and at 365 days from stroke onset. Good functional outcome was defined as mRS 0–2. We identified factors associated with mortality and a good functional outcome using univariate logistic regression analysis, with statistically significant variables subsequently included into multivariate analyses.

Results. Among 593 MT-treated AIS patients, AKI was found in 12.6%. Patients with AKI had significantly higher mortality and worse functional outcome at discharge, and at 90, and at 365 days from stroke onset. AKI was an independent factor associated with mortality and worse functional outcome at discharge, and at 90, and at 365 days from stroke onset. AKI remained independently associated with a lower chance of a good functional outcome in a 365-day follow-up when the analysis was limited to patients who survived until discharge (OR = 0.244, 95% CI: 0.095–0.624, p = 0.003).

Conclusions and clinical implications. AKI during hospitalisation is an independent risk factor of short- and long-term mortality and poor functional outcome in patients with AIS undergoing MT. There is a need to create a protocol to monitor kidney function and ensure prompt AKI treatment in MT-treated AIS patients.

Keywords. acute kidney injury, acute ischaemic stroke, mechanical thrombectomy, stroke outcomes, endovascular stroke treatment

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Introduction

Acute kidney injury (AKI) is defined as an abrupt worsening of kidney function. It represents a heterogenic phenomenon with multiple potential causes and a broad clinical spectrum. According to the KDIGO (Kidney Disease Improving Global Outcomes) guidelines, a diagnosis of AKI requires either an increase of serum creatinine level of at least 26 μmol/L within 48 hours, or at least 1.5 times its baseline value within seven days, or urine output of less than 0.5mL/kg/h for six hours [1]. AKI in hospitalised patients has been associated with high rates of in-hospital mortality [2] and an increased risk of death in long-term follow-up [3].

AKI is a relatively common problem in patients with acute ischaemic stroke (AIS), being reported, according to different studies, in 8–21% of AIS cases [4]. AKI in stroke patients has been associated with higher mortality and a worse functional outcome [5–7]. Potential causes of AKI developing in AIS patients include infections, hypovolemia, the use of nephrotoxic medications, and renal hypoperfusion [8]. It can also be related to the use of iodine contrast agents and catheter-based procedures. These are inevitable if the patient is to be treated with mechanical thrombectomy (MT), which is the optimal treatment of AIS caused by large vessel occlusions (LVO). MT also involves intra-arterial contrast administration, something that seems to be associated with a higher risk of AKI than when administered intravenously [9].

Clinical rationale for study

The literature regarding the impact of AKI on the prognosis for MT-treated AIS patients is scarce, especially concerning long-term outcomes. A recent systematic review with meta-analysis showed a pooled incidence of 7% of AKI among AIS patients treated with MT, and one of the few studies to have examined its influence on MT outcomes showed its association with a higher risk of death and dependency in a 3-month follow-up [10].

The aim of this study was to assess the impact of AKI during hospitalisation on short- and long-term outcomes of MT-treated AIS patients, up to 365 days after stroke onset.

Material and methods

This was an observational cohort study taking the form of a retrospective analysis of data prospectively collected as a part of the 'Identification and clinical validation of biomarkers for long-term outcome after cerebral ischaemia (IBioStroke)' study (https://www.neuron-eranet.eu/wp-content/uploads/iBioStroke.pdf). Into the analysis we included all AIS patients treated with MT in the Comprehensive Stroke Centre (CSC) of the University Hospital in Krakow, Poland from 2019 to 2021.

We collected information during hospitalisation concerning patient age, biological sex, the profile of cardiovascular risk factors known at stroke onset (*i.e.* arterial hypertension, atrial fibrillation, diabetes mellitus, history of stroke/TIA, hypercholesterolaemia, history of myocardial infarction, obesity, history of smoking, alcohol abuse, chronic kidney disease) and the presence of carotid artery atherosclerosis (assessed via ultrasound). We assessed the neurological deficit at admission using the National Institutes of Health Stroke Scale (NIHSS). We estimated penumbra and infarct volumes based on computed tomography (CT) perfusion analysis using an iRAPID post-processing program. We noted whether the patient was concomitantly treated with intravenous thrombolysis (IVT). We also noted time from stroke onset to groin puncture and the radiological effect of the treatment assessed with a modified Treatment in Cerebral Ischaemia (mTICI) scale, with successful reperfusion defined as an mTICI score of 2B-3. A secondary intracranial haemorrhage after procedure (ICH) and a need for Intensive Care Unit (ICU) treatment were also noted.

We identified patients who developed acute kidney injury (AKI) during hospitalisation, based on available serum creatinine (SCr) level test results. AKI was defined as a rise in SCR of ≥ 26.5 μmol/L within 48 hours, or a ≥ 1.5-fold SCr increase within seven days, observed during hospitalisation. Factors associated with the occurrence of AKI in this group were those identified in our previous study [11].

Short term outcome was defined as in-hospital mortality, discharge neurological deficit assessed using NIHSS scale, the ability to perform activities of daily living assessed using Barthel index, and neurological functional outcome assessed using a modified Rankin scale (mRS), with good functional outcome being defined as an mRS score of 0–2. In patients in whom long term-follow up was available, we noted the mortality and neurological functional outcome assessed using mRS at 90 and at 365 days from stroke onset. The mRS score after discharge was obtained during visits to the outpatient clinic of the Krakow CSC or through telephone interviews with patients or their caregivers.

We compared the short-and long-term outcomes between groups of patients with and without AKI during hospitalisation. We presented categorical data as counts and percentages and compared it using a Chi-square test. Continuous data distribution was assessed using a Kolmogorov-Smirnov test and, where there was non-normal data distribution, compared using a U-Mann Whitney test. Two-tailed p-value of < 0.05 was considered statistically significant. We subsequently assessed which factors contributed to in-hospital, 90-day and 365-day functional outcome and mortality using univariate logistic regression analyses, followed by multivariable analyses including statistically significant variables. In cases when the number of variables in the multivariate analysis was too high for our sample size, or resulted in a poor-fit model, we excluded an adequate number of variables with the highest p-value identified during univariate analysis. All statistical analyses were performed using a PS Imago Pro 9.0 program.

Our report was prepared according to STROBE (Strengthening the reporting of observational studies in epidemiology) guidelines [12]. The study was 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) and Jagiellonian University grant number N41/DBS/001270. This study had Jagiellonian University Bioethics Committee approval (decision number 1072.6120.118.2020).

Results

The study included 593 MT-treated AIS patients aged 18–97 with a median age of 70 (IQR = 17), of whom 311 (52.4%) were male. AKI criteria were fulfilled by 75 patients (12.6%). The highest creatinine levels in the AKI subgroup were detected on days 2–31 of hospitalisation, median 6 days (IQR = 3–12). In 42 AKI patients (56%), the peak creatinine level was found during the first seven days of hospitalisation. Factors associated with the occurrence of AKI were as described in our previous study [11]. iRAPID CT perfusion analysis was available in 529/593 patients (89.2%).

There were no statistically significant differences between groups of patients with and without AKI concerning age or biological sex. Patients with AKI compared to those without AKI significantly more often needed treatment in the ICU (29.3% vs. 6.8%, $p < 0.001$). Short-term outcomes were significantly worse in the AKI (+) group, which had much higher in-hospital mortality (52.0% vs. 8.1%, $p < 0.001$), as well as, for patients who survived, higher median NIHSS score at discharge [10 (IQR = 15) vs. 4 (IQR = 9), $p < 0.001$], much lower Barthel index score at discharge [0 (IQR = 36) vs. 70 (IQR = 80), $p < 0.001$], and much lower rate of good functional outcome at discharge (14.7% vs. 53.5%, $p < 0.001$). The causes of in-hospital mortality in AKI (+) group are set out in Table 1.

90-day follow-up was available in 578 patients (97.5%) and the outcomes were significantly worse in AKI (+) patients, who had a much higher mortality rate (66.7% vs. 16.5%, $p < 0.001$) and a much lower percentage of good functional outcome (16.0% vs. 62.8%, $p < 0.001$).

365-day follow-up was available in 557 patients (93.9%), and the outcomes were also significantly worse in the AKI (+) group, with a much higher mortality rate (74.6% vs. 24.7%, $p < 0.001$) and a good functional outcome achieved much less frequently (16.9% vs. 66.5%, $p < 0.001$). The abovementioned results are set out in Table 2.

Multivariate analysis showed that AKI was an independent factor associated with mortality during hospitalisation (OR = 13.831, 95% CI: 7.330-32.294, $p < 0.001$), in 90-day observation (OR = 15.912, 95% CI: 7.615–33.247, $p < 0.001$), as well as in 365-day follow-up (OR = 12.380, 95% CI: 5.891–26.019, $p < 0.001$). AKI during hospitalisation was also independently associated with a lower chance of good functional outcome at discharge (OR = 0.120, 95% CI: 0.053–0.273, $p < 0.001$), at 90 days (OR = 0.113, 95% CI: 0.051–0.251, $p < 0.001$), and at 365 days (OR = 0.093, 95% CI: 0.042–0.207, $p < 0.001$). Multivariate logistic regression analysis results are set out in detail in Table 3.

Table 1. Causes of in-hospital mortality in AIS patients treated with MT who developed AKI during hospitalisation

Cause	Number of patients
Multiple organ dysfunction	27 (69.2%)
Brain death	4 (10.3%)
Brain oedema and herniation	3 (7.7%)
Pneumonia and respiratory failure	2 (5.1%)
Acute hepatic failure	2 (5.1%)
Sudden cardiac arrest of an unknown cause	1 (2.6%)

AIS — acute ischaemic stroke; MT — mechanical thrombectomy; AKI — acute kidney injury

Table 2. Comparison of groups of patients with and without AKI during hospitalisation

	AKI (+)	AKI (–)	P-value
NIHSS score on admission [median (IQR)]	16 (IQR = 10)	16 (IQR = 9)	0.273
Short-term outcome (n = 593)			
ICU admission [n (%)]	22 (29.3%)	35 (6.8%)	< 0.001
NIHSS score at discharge [median (IQR)]	10 (IQR = 15)	4 (IQR = 9)	< 0.001
Barthel index score at discharge [median (IQR)]	0 (IQR = 36)	70 (IQR = 80)	< 0.001
Good functional outcome at discharge [n (%)]	11 (14.7%)	277 (53.5%)	< 0.001
In-hospital mortality [n (%)]	39 (52.0%)	42 (8.1%)	< 0.001
90-day outcome (n = 578)			
Mortality [n (%)]	50 (66.7%)	83 (16.5%)	< 0.001
Good functional outcome [n (%)]	12 (16.0%)	316 (62.8%)	< 0.001
365-day outcome (n = 557)			
Mortality [n (%)]	53 (74.6%)	120 (24.7%)	< 0.001
Good functional outcome [n (%)]	12 (16.9%)	323 (66.5%)	< 0.001

AKI — acute kidney injury; ICU — intensive care unit; NIHSS — National Institutes of Health Stroke Scale; Good functional outcome — modified Rankin scale score of 0–2 points

Table 3. Multivariate analysis of factors associated with short- and long-term outcomes (in-hospital, 90-day, and 365-day mortality and good functional outcome) after mechanical thrombectomy. Factors included in table were identified using univariate logistic regression analysis. In cases when number of variables in multivariate analysis was too high for our sample size, or resulted in a poor-fit model, we excluded some variables with highest p-values identified during univariate analysis

In-hospital mortality (Nagelkerke R2 = 0.361)			
History of stroke/TIA	p = 0.304	OR = 1.547	95% CI: 0.673–3.555
NIHSS score at admission [points]	p < 0.001	OR = 1.121	95% CI: 1.063–1.183
Infarct volume at admission [mL]	p = 0.067	OR = 1.008	95% CI: 0.999–1.016
Intravenous thrombolysis	p = 0.018	OR = 0.464	95% CI: 0.246–0.874
AKI	p < 0.001	OR = 13.831	95% CI: 6.850–27.926
ICH	p = 0.007	OR = 2.467	95% CI: 1.276–4.769
Good functional outcome at discharge (Nagelkerke R2 = 0.403)			
Age [years]	p = 0.011	OR = 0.978	95% CI: 0.961–0.995
History of smoking	p = 0.014	OR = 2.004	95% CI: 1.154–3.481
NIHSS score at admission [points]	p < 0.001	OR = 0.886	95% CI: 0.851–0.921
Penumbra volume at admission [mL]	p < 0.014	OR = 0.996	95% CI: 0.993–0.999
Infarct volume at admission [mL]	p < 0.001	OR = 0.984	95% CI: 0.976–0.993
Full reperfusion after MT	p < 0.001	OR = 3.994	95% CI: 1.996–7.992
AKI	p < 0.001	OR = 0.120	95% CI: 0.053–0.273
ICH	p = 0.001	OR = 0.428	95% CI: 0.257–0.714
90-day mortality (Nagelkerke R2 = 0.428)			
Age [years]	p < 0.001	OR = 1.049	95% CI: 1.024–1.074
History of smoking	p = 0.925	OR = 0.965	95% CI: 0.460–2.025
Carotid artery atherosclerosis	p = 0.033	OR = 0.399	95% CI: 0.171–0.930
Chronic kidney disease	p = 0.229	OR = 1.578	95% CI: 0.750–3.321
NIHSS score at admission [points]	p < 0.001	OR = 1.110	95% CI: 1.057–1.165
Penumbra volume at admission [mL]	p = 0.119	OR = 1.003	95% CI: 0.999–1.006
Infarct volume at admission [mL]	p = 0.002	OR = 1.012	95% CI: 1.005–1.020
Full reperfusion after MT	p = 0.002	OR = 0.336	95% CI: 0.169–0.669
AKI	p < 0.001	OR = 15.912	95% CI: 7.615–33.247
ICH	p = 0.004	OR = 2.300	95% CI: 1.311–4.035
90-day good functional outcome (Nagelkerke R2 = 0.409)			
Age	p < 0.001	OR = 0.964	95% CI: 0.946–0.982
History of stroke / TIA	p = 0.158	OR = 0.617	95% CI: 0.316–1.206
History of smoking	p = 0.271	OR = 1.381	95% CI: 0.777–2.453
Chronic kidney disease	p = 0.072	OR = 0.505	95% CI: 0.240–1.063
NIHSS score at admission [points]	p < 0.001	OR = 0.908	95% CI: 0.873–0.946
Penumbra volume at admission [mL]	p = 0.145	OR = 0.998	95% CI: 0.995–1.001
Infarct volume at admission [mL]	p < 0.001	OR =0.984	95% CI: 0.975–0.992
Intravenous thrombolysis	p = 0.002	OR = 2.052	95% CI: 1.305–3.228
Full reperfusion after MT	p < 0.001	OR = 3.819	95% CI: 1.979–7.371
AKI	p < 0.001	OR = 0.113	95% CI: 0.051–0.251
ICH	p = 0.006	OR = 0.486	95% CI: 0.289–0.817
365-day mortality (Nagelkerke R2 = 0.388)			
Age [years]	p < 0.001	OR = 1.043	95% CI: 1.022–1.065
History of smoking	p = 0.446	OR = 0.776	95% CI: 0.404–1.489
Carotid artery atherosclerosis	p = 0.166	OR = 0.623	95% CI: 0.319–1.218
Chronic kidney disease	p = 0.198	OR = 1.596	95% CI: 0.783–3.251
NIHSS score at admission [points]	p < 0.001	OR = 1.082	95% CI: 1.038–1.128

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Table 3 cont. Multivariate analysis of factors associated with short- and long-term outcomes (in-hospital, 90-day, and 365-day mortality and good functional outcome) after mechanical thrombectomy. Factors included in table were identified using univariate logistic regression analysis. In cases when number of variables in multivariate analysis was too high for our sample size, or resulted in a poor-fit model, we excluded some variables with highest p-values identified during univariate analysis

Penumbra volume at admission [mL]	p = 0.038	OR = 1.003	95% CI: 1.000–1.007
Infarct volume at admission [mL]	p = 0.056	OR = 1.007	95% CI: 1.000–1.014
Intravenous thrombolysis	p = 0.001	OR = 0.460	95% CI: 0.286–0.740
Full reperfusion after MT	p = 0.002	OR = 0.353	95% CI: 0.185–0.674
AKI	p < 0.001	OR = 12.380	95% CI: 5.891–26.019
ICH	p = 0.007	OR = 2.079	95% CI: 1.225–3.527
365-day good functional outcome (Nagelkerke R2 = 0.409)			
Age [years]	p < 0.001	OR = 0.953	95% CI: 0.934–0.972
History of smoking	p = 0.833	OR = 1.066	95% CI: 0.590–1.924
Chronic kidney disease	p = 0.057	OR = 0.492	95% CI: 0.237–1.021
NIHSS score at admission [points]	p < 0.001	OR = 0.918	95% CI: 0.882–0.955
Penumbra volume at admission [mL]	p = 0.087	OR = 0.997	95% CI: 0.994–1.000
Infarct volume at admission [mL]	p < 0.001	OR = 0.987	95% CI: 0.979–0.994
Intravenous thrombolysis	p < 0.001	OR = 2.176	95% CI: 1.373–3.448
Full reperfusion after MT	p < 0.001	OR = 4.008	95% CI: 2.084–7.708
AKI	p < 0.001	OR = 0.093	95% CI: 0.042–0.207
ICH	p = 0.017	OR = 0.531	95% CI: 0.316–0.894

TIA — transient ischaemic attack; NIHSS — National Institutes of Health Stroke Scale; AKI — acute kidney injury; ICH — intracerebral haemorrhage; MT — mechanical thrombectomy; Good functional outcome — modified Rankin Scale score of 0–2 points; Full reperfusion — modified Treatment in Cerebral Ischaemia scale result of 2b-3

Table 4. Multivariate analyses showing factors significantly associated with good functional outcome 365 days after mechanical thrombectomy in patients who survived until discharge

365-day good functional outcome (Nagelkerke R2 = 0.302)			
Age [years]	p < 0.001	OR = 0.956	95% CI: 0.935–0.978
Female sex	p = 0.051	OR = 0.594	95% CI: 0.351–1.003
Diabetes	p = 0.107	OR = 0.615	95% CI: 0.341–1.110
History of smoking	p = 0.806	OR = 0.921	95% CI: 0.476–1.780
Chronic kidney disease	p = 0.118	OR = 0.533	95% CI: 0.242–1.173
NIHSS score at admission [points]	p = 0.002	OR = 0.935	95% CI: 0.896–0.977
Penumbra volume at admission [ml]	p = 0.050	OR = 0.997	95% CI: 0.993–1.000
Infarct volume at admission [ml]	p = 0.001	OR = 0.987	95% CI: 0.979–0.995
Full reperfusion after MT	p < 0.001	OR = 4.044	95% CI: 2.011–8.134
AKI	p = 0.003	OR = 0.244	95% CI: 0.095–0.624
sICH	p = 0.110	OR = 0.625	95% CI: 0.351–1.112

NIHSS — National Institutes of Health Stroke Scale; AKI — acute kidney injury; sICH — secondary intracerebral haemorrhage; MT — mechanical thrombectomy; Good functional outcome — modified Rankin Scale score of 0–2 points; Full reperfusion — modified Treatment in Cerebral Ischaemia scale result of 2b-3

AKI remained independently associated with a lower chance of a good functional outcome 365 days after stroke (OR = 0.244, 95% CI: 0.095–0.624, p = 0.003), and also when the analysis was limited only to 476 patients who survived until discharge and in whom a long-term follow-up was available. The multivariate analysis results in this group are set out in Table 4. AKI was also associated with 365-day mortality in patients who survived until discharge (OR = 3.650, 95%CI: 1.741–7.649, p < 0.001) in univariate analysis, but the number of patients with AKI who died between discharge and 365 days after stroke was too small to allow multivariate analysis.

Discussion

Our study showed that AKI during hospitalisation is an independent factor associated with mortality and worse functional outcome of MT-treated AIS patients up to 365 days after stroke onset. To the best of our knowledge, this is the first study to assess the impact of AKI during hospitalisation on the prognosis for patients undergoing MT in such a long-term follow-up. Previous studies on this topic have reported outcomes up to three months after stroke onset, and their results also pointed towards a worse prognosis in patients with AKI.

Weber et al. reported increased in-hospital mortality in MT-treated AIS patients developing AKI [13]. Alqahtani et al. identified AKI to be an independent predictor of a poor functional outcome, defined as in-hospital death or discharge to a hospice [14]. In a study by Laible et al., post-contrast AKI was independently associated with mortality, both in-hospital and at 3-month follow-up [15]. A study by Diprose et al. showed that contrast-associated AKI was an independent factor associated with mortality at 3-month follow-up [16]. Fandler-Höfler et al. found AKI during hospitalisation to be an independent factor associated with a poor functional outcome in a 3-month observation [17]. Finally, in a study by Yoo et al., AKI was associated with increased mortality and a lower percentage of good functional outcomes in a 3-month follow-up [18].

The studies just mentioned used different definitions of AKI. In the presented paper, we diagnosed AKI based on KDIGO guidelines, but limited our analysis to SCr, as it was impossible to analyse urine output retrospectively. We decided to only analyse SCr results obtained during hospitalisation in our Department — this was due to the fact that the patients had been transferred for MT either from other hospitals (the 'drip and ship' strategy, applying to c.50% of our group), where laboratory methods and norms may have been different, or from the Accident & Emergency Department, where SCr level is assessed using point-of-care testing, meaning that it cannot be directly compared to results obtained in a laboratory. Although clinicians were aware of the kidney parameters before admission, we decided not to analyse them, for the abovementioned reasons. A first SCr result was available in most patients only after treatment with MT (but, in such cases, usually within 24 hours of admission).

This is the most important limitation of our study, and could potentially have led to some AKI cases going unrecognised. Nevertheless, a study by Arnold et al. showed the incidence of AKI to be similar regardless of whether a pre-admission or a first-during-admission SCr level was used, although using results from after the admission led to a risk of underestimating 30-day and 365-day mortality [5]. This may suggest that the negative effect of AKI during hospitalisation on MT treatment prognosis could be even higher than presented in this study. We also decided not to consider the best SCr result during hospitalisation as the baseline, because, even if it was accurate, we did not have any data on the dynamics of kidney function decline before admission.

Other limitations of our study included its single-centre and retrospective character. Serum creatinine levels were not routinely assessed every day, which may have influenced the detectability of AKI in our group. Direct AKI aetiology was hard to assess retrospectively, especially given that in many cases more than one potential cause for the development of AKI could be identified in a single patient.

The incidence of AKI in AIS patients treated with MT has previously been estimated to be c.7% [10]. Our study showed a higher occurrence of 12.6%, and this difference probably resulted from differing methodologies and definitions of AKI in different studies. The factors most frequently reported to be associated with AKI after MT are diabetes mellitus, impaired baseline renal function, and higher contrast volume administration [10]. Concomitant COVID-19 infection also makes MT-treated AIS patients more prone to developing AKI [19]. In our group, the percentages of patients with diabetes and chronic kidney disease were 20.1% and 9.9%, respectively.

Clinical implications / future directions

Further, preferably multicentre, studies should be performed to establish the importance of AKI impact on stroke prognosis, among other factors that so far have been associated with outcomes of AIS patients [20], especially those undergoing endovascular stroke therapy [21, 22]. Clinicians should identify patients at risk of AKI and carefully monitor their renal function. As AKI is potentially treatable and, in many cases, preventable, the results of this study should encourage stroke specialists to pay close attention to the risk factors of AKI present in their patients and to develop protocols for AKI diagnosis and prompt treatment. Although it is impossible to perform MT without the use of contrast, some precautions can still be made to protect patients from developing AKI [23]. These include identifying patients with a high risk of AKI, adequate patient hydration, avoiding nephrotoxic medications, optimising blood pressure, and, if possible, avoiding further contrast exposure after the procedure [24].

Article information

Data availability statement: *The data supporting the findings of this study is available from the corresponding author upon reasonable request.*

Ethics statement: *This study had Jagiellonian University Bioethics Committee approval (decision number 1072.6120.118.2020).*

Authors' contributions: *KS: conceptualisation, statistical analysis, draft writing and editing; PW: major role in data acquisition, draft editing; KZ, DW, PS: data acquisition; KW:*

statistical analysis; TP: data acquisition, draft editing; AS, MK: conceptualisation, draft editing, supervision.

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