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Emergent CT angiography and risk of contrast-induced acute kidney injury in acute ischaemic stroke

Angiografia TK wykonywana w trybie pilnym a ryzyko ostrego uszkodzenia nerek wywołanego podaniem środka kontrastującego u pacjentów z ostrym udarem niedokrwiennym mózgu

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Abstract

Objectives: Emergent computed tomography angiography with contrast is commonly performed for acute ischaemic stroke, but potentially delayed pending admission renal function assessment due to purported risk of contrast-induced acute kidney injury. Such clinical dilemma warrants further evaluation. **Methods:** We retrospectively examined the incidence of contrast-induced acute kidney injury in acute ischaemic stroke patients who underwent a single initial contrasted computed tomography angiography or two serial contrasted computed tomography angiographies, versus acute kidney injury in patients with no contrast exposure. Acute kidney injury and extended renal dysfunction were defined as increase by >50% in serum creatinine from admission, within 5 days and after 30 days respectively. **Results:** Of 465 patients with acute ischaemic stroke, 372 underwent computed tomography angiography (203 with single initial contrasted computed tomography angiography, 169 with two serial contrasted computed tomography angiographies), and 93 patients had no contrast exposure. 33% of entire cohort had diabetes mellitus and 9.4% had chronic kidney disease, both comparable between subgroups. Acute kidney injury occurred in 2.5%, 2.4%, and 9.7% with single initial contrasted computed tomography angiography, two serial contrasted computed tomography angiographies, and no contrast exposure, respectively ($p = 0.004$). Corresponding rates of extended renal dysfunction were 1.5%, 0.6%, and 6.5% ($p = 0.185$). On multivariate analysis, diabetes mellitus and lower baseline estimated glomerular filtration rate were independently associated with acute kidney injury, while lower estimated glomerular filtration rate was associated with extended renal dysfunction ($p < 0.05$). Contrast-exposed patients did not have higher risk for acute kidney injury (odds ratio, OR = 0.25, 95% CI 0.096–0.647, $p = 0.004$) or extended renal dysfunction (OR = 0.083, 95% CI 0.008–0.810, $p = 0.032$) versus non-contrasted patients. Receiving two computed tomography angiographies within 24 hours did not confer added risk for contrast-induced acute kidney injury. **Conclusions:** Emergent or serial computed tomography angiographies in acute ischaemic stroke were associated with very low risk of acute kidney injury and extended renal dysfunction, and these risks were not significantly higher than in acute ischaemic stroke patients with no early contrast exposure.

Key words: acute kidney injury, cerebral infarction, computed tomography, contrast media, stroke, thrombolytic therapy

Streszczenie

Cel pracy: Angiografię metodą tomografii komputerowej z podaniem kontrastu (angio-TK) powszechnie stosuje się w przypadku ostrego niedokrwiennego udaru mózgu, jednakże często wykonanie badania jest opóźniane w celu dokonania uprzedniej oceny funkcji nerek w związku z domniemanym ryzykiem wystąpienia ostrego uszkodzenia nerek indukowanego kontrastem radiologicznym. Ten istniejący w praktyce klinicznej dylemat zasługuje na bliższe zbadanie w celu rozwiania wątpliwości dotyczących należytego sposobu postępowania. **Metoda:** Przeprowadziliśmy retrospektywną analizę częstości występowania ostrego uszkodzenia nerek spowodowanego podaniem kontrastu wśród pacjentów z ostrym udarem niedokrwiennym mózgu, u których wykonano pojedyncze wstępne badanie angio-TK lub dwa następujące po sobie badania angio-TK, w porównaniu z częstością występowania ostrego uszkodzenia nerek u pacjentów bez ekspozycji na kontrast. Ostre uszkodzenie nerek i przedłużająca się niewydolność nerek zostały zdefiniowane jako wzrost stężenia kreatyniny

w surowicy o ponad 50% w stosunku do stężenia obecnego w momencie przyjęcia pacjenta odpowiednio w okresie 5 i 30 dni od daty przyjęcia. **Wyniki:** Spośród 465 pacjentów z ostrym udarem niedokrwiennym mózgu u 372 wykonano angio-TK (u 203 pacjentów pojedyncze wstępne badanie angio-TK, a u 169 dwa następujące po sobie badania angio-TK), natomiast 93 pacjentom nie podawano kontrastu. U 33% pacjentów z całej kohorty występowała cukrzyca, a u 9,4% przewlekła choroba nerek, przy czym odsetki te kształtowały się podobnie w obu podgrupach. Ostre uszkodzenie nerek wystąpiło u 2,5% pacjentów, u których przeprowadzono pojedyncze badanie angio-TK, u 2,4% pacjentów, którzy przeszli dwa następujące po sobie badania angio-TK, oraz u 9,7% pacjentów bez ekspozycji na kontrast ($p = 0{,}004$). Odsetek pacjentów z przedłużającą się niewydolnością nerek wyniósł w tych samych grupach odpowiednio 1,5% (pojedyncze badanie angio-TK), 0,6% (dwa następujące po sobie badania angio-TK) oraz 6,5% (brak badania angio-TK) ($p = 0{,}185$). Analiza wielowariantowa wykazała, iż cukrzyca oraz niższy wyjściowy szacunkowy współczynnik przesączania kłębuszkowego były w sposób niezależny związane z przedłużającą się niewydolnością nerek ($p < 0{,}05$), natomiast niższy współczynnik przesączania kłębuszkowego wiązał się z przedłużającą się niewydolnością nerek. U pacjentów, którym podano środek kontrastujący, nie występowało zwiększone ryzyko ostrego uszkodzenia nerek (OR = 0,25, 95% CI 0,096–0,647, $p = 0{,}004$) ani też przedłużającej się niewydolności nerek (OR = 0,083, 95% CI 0,008–0,810, $p = 0{,}032$) w porównaniu z pacjentami, którym nie podawano środka kontrastującego. Przeprowadzenie u pacjenta dwóch następujących po sobie badań angio-TK w 24-godzinnym przedziale czasowym nie zwiększało ryzyka ostrego uszkodzenia nerek wywołanego ekspozycją na środek kontrastujący. **Wnioski:** Zarówno pojedyncze, jak i powtarzane badania angio-TK wykonywane u pacjentów z ostrym udarem niedokrwiennym mózgu związane były z bardzo niskim ryzykiem ostrego uszkodzenia nerek i przedłużającej się niewydolności nerek, a ryzyko to nie było istotnie statystycznie wyższe niż u pacjentów z ostrym udarem mózgu, u których nie przeprowadzano wczesnego badania z podaniem środka kontrastującego.

Słowa kluczowe: ostra niewydolność nerek, udar niedokrwienny mózgu, tomografia komputerowa, środki kontrastujące, leki trombolityczne

INTRODUCTION

Stroke is one of the leading causes of patient mortality and morbidity (Rosamond *et al.*, 2008). Patients who present within 3 to 4.5 hours of the symptom-onset in acute ischaemic stroke (AIS) are considered for intravenous tissue plasminogen activator (tPA) which remains the only approved therapeutic agent for achieving arterial recanalization (Tissue plasminogen activator for acute ischaemic stroke, 1995). Prior to treatment, most patients will undergo imaging of the brain of which computed tomography (CT) is the fastest and most cost-effective.

Multimodal CT-based imaging in AIS includes CT-angiography (CTA) and CT-perfusion. They provide rapid information about intracranial arterial occlusion, status of various collaterals and cerebral hemodynamics that might influence therapeutic decision-making. CTA as an imaging modality in AIS has many advantages including rapid accessibility, low cost, short scanning-time interval and patient tolerability (Lev *et al.*, 2001; Smith *et al.*, 2003). However, widespread use of CT-based multimodal imaging has been constrained by concerns about potential nephrotoxicity, particularly because time pressure in evaluating patients with AIS can necessitate making decisions regarding contrast administration *in the absence* of an admission serum creatinine (sCr) value. With the success of mechanical thrombectomy in large vessel occlusion (Berkhemer *et al.*, 2015; Campbell *et al.*, 2015; Goyal *et al.*, 2015; Jovin *et al.*, 2015; Saver *et al.*, 2015) it is now imperative that a CTA is performed in all acute stroke patients when there is access to such therapeutic options. It is equally important that the CTA does not put the patient at an unacceptable risk of renal dysfunction from contrast exposure.

Smith *et al.* demonstrated that waiting for the admission sCr resulted in lengthy delay in the initiation of thrombolytic therapy in AIS (Smith *et al.*, 2003), thus reducing the chances of a good functional outcome (Lees *et al.*, 2010). Furthermore, administration of a radio-contrast agent in patients without the knowledge of pre-scan kidney function might pose a specific risk of further acute kidney injury (AKI), especially in patients with pre-existing chronic kidney disease (CKD), and it is important to quantify this risk for informed consent.

Contrast-induced acute kidney injury (CI-AKI) is generally regarded as a transient nephropathy that develops after angiographic procedures using contrast media. Nonetheless, CI-AKI accounts for a substantial proportion of hospital-acquired AKI (Nash *et al.*, 2002). CI-AKI is associated with an increased risk of extended renal dysfunction and cardiovascular morbidity (Best *et al.*, 2002; Chua *et al.*, 2014; Gruberg *et al.*, 2000; McCullough *et al.*, 2006; Nash *et al.*, 2002); however, the acute stroke patient is also at risk of a lifetime of dependence. It is difficult to determine whether the benefits of emergent CTA outweigh the risk of worsened acute or extended renal outcomes. Therefore, we examined the incidence of CI-AKI among patients with AIS who underwent emergent CTA versus AKI in patients not exposed to radio-contrast. We hypothesized that (i) emergent CTA would not confer a higher risk of AKI or extended renal dysfunction compared with patients without radio-contrast exposure, and (ii) two CTA performed within 24 hours in an AIS patient is not associated with adverse renal outcomes compared with single CTA.

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METHODS

Study design and setting

We conducted a single-centre retrospective observational study, in an 1160-bed tertiary institution with facilities and protocol for emergent thrombolysis, and examined the incidence of AKI in AIS patients with versus without CTA and contrast exposure. Consecutive AIS patients presenting within 4.5 hours of symptom-onset between January 2007 and October 2013 were enrolled in this study. Our Human Research Ethics Committee approved this study and waived the need for informed consent (DSRB 2010/00509).

Participants and comparators

Potential study subjects were identified from our hospital acute stroke database. AIS patients aged >18 years who presented within 4.5 hours of symptom-onset were included in this study. All patients had admission sCr measured in the emergency department. sCr was measured again within 5 days of hospitalization. We collected information on further sCr estimations, if they were performed within 30–120 days of the stroke-onset. Patients without follow-up sCr estimation (within 5 days of stroke-onset) were excluded. We excluded patients with pre-existing end-stage renal disease already on renal replacement therapy. sCr measurements were done on Advia Centaur 2400, using enzymatic method traceable to the IDMS Reference Method via correlation of patient samples and reference material SRM967 from the National Institute of Standards and Technology (NIST). We compared the acute and extended renal outcomes in patients who underwent a single initial CTA (contrast-exposed with 1 CTA scan – CE1), patients who had 2 serial CTAs (contrast-exposed with 2 serial CTA scans – CE2), and with patients who did not receive any exposure to radio-contrast (scan with no contrast-exposure – NCE). The decision to perform the CTA or a non-contrasted CT was at the discretion of doctors in the emergency department.

Outcome measures

The primary outcome was AKI, as defined by a relative increase of >50% in sCr from the admission value within 5 days of admission [Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012], which was presumed contrast-related (CI-AKI) in patients exposed to radio-contrast for CTA. This was compared with AKI from alternative precipitants in the AIS patients who did not receive a radio-contrast agent. Secondary outcomes included transient renal dysfunction that referred to initial AKI that resolved within 30 days, whereas extended renal dysfunction was defined as persistently elevated sCr after 30 days. Delta-sCr was defined as absolute difference in subsequent sCr from admission value.

CTA protocol

Non-enhanced CT and high-resolution CTA acquisitions were performed according to standard departmental protocols with a 64-slice multi-detector helical scanner (Philips Inc, US), and images were acquired with 70 mL bolus injection of contrast. Scan parameters at our institution were: slice thickness 1 mm; no slice gap; field of view 200 mm; matrix 512 × 512 and 230–250 mAs. Coverage was from the base of skull to the vertex and the source images were reformatted into 3 mm-thick axial, coronal and sagittal projections.

Data analysis

As baseline chronic kidney disease (CKD) is an important predictor of AKI (Mehran and Nikolsky, 2006), estimated glomerular filtration rate (eGFR) was calculated from the admission sCr (in μmol/L) and demographics using the abbreviated 4-variable MDRD equation (Levey *et al.*, 2000):

$$eGFR = 186 \times (sCr/88.4)^{-1.154} \times (Age)^{-0.203} (\times 0.742 \text{ if female}) (\times 1.210 \text{ if black})$$

We performed univariate comparison of study variables between the CE group ($n = 372$) and the NCE group ($n = 93$). Continuous variables are reported as mean (standard deviation) or median (interquartile range) where appropriate for the data distribution, and analysed using parametric or non-parametric tests, respectively. Categorical variables are presented in frequency (percentage) and compared using Chi-square test. A multivariate logistic regression model was used to compare the risk of AKI between the contrast-enhanced and non-contrasted group after adjusting for possible confounders. A 2-tailed p-value of <0.05 is taken as measure of statistical significance. The statistical software used was SPSS version 20 (IBM, Armonk, NY, USA).

RESULTS

Four hundred and sixty-five patients fulfilled the study criteria and were included for analysis. Of these, 203 were of CE1, 169 CE2, and 93 patients with NCE that served as controls (Fig. 1). Of the 465 patients, the median age was 65 (18–92) years. One hundred and fifty-four (33%) suffered from diabetes mellitus and 44 (9%) patients had baseline CKD with eGFR <60 mL/min/1.73 m^2, and these were comparable among all subgroups. The median admission sCr was 85 μmol/L (31–744).

Eighteen (4%) patients developed AKI. No patients required renal replacement therapy. Five of 203 patients (2.5%) with CE1 developed CI-AKI. Four of 169 patients (2.4%) with CE2 developed CI-AKI. AKI developed in 9 of 93 patients (9.7%) with NCE ($p = 0.004$, Tab. 1).

On univariate analysis, patients with diabetes mellitus, lower baseline eGFR, and NCE had higher risk of AKI.

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On multivariate analysis, only diabetes mellitus and lower baseline eGFR were independently associated with AKI ($p < 0.05$, Tab. 2). Contrast-enhanced imaging did not confer a higher risk of AKI or extended renal dysfunction (Tab. 2).

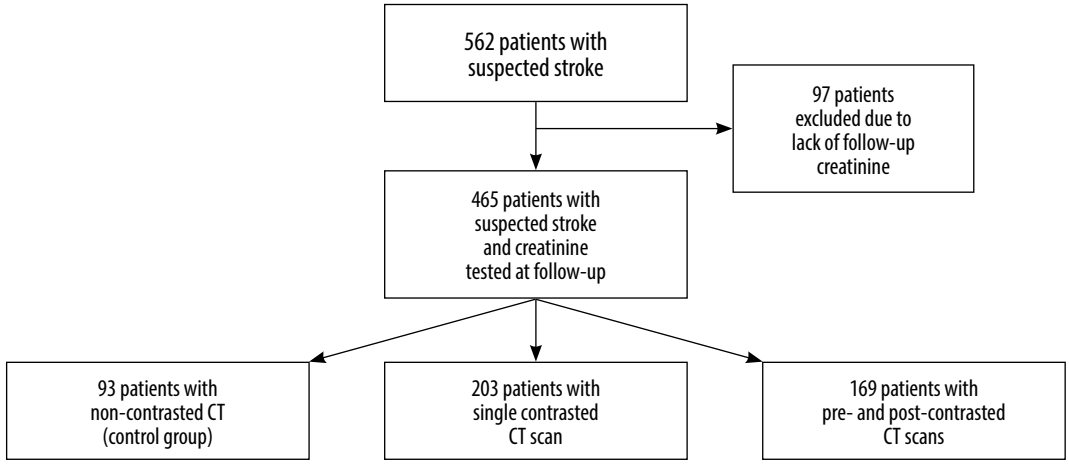
Median delta-sCr for the total cohort was a decrease of 11 µmol/L (–75 to 285 µmol/L) in the first 5 days and a decrease of 2.5 µmol (–65 to 351 µmol/L) at 30 days. Using the delta-sCr in NCE patients at 5 and 30 days as reference, the delta-sCr in CE1 or CE2 patients were not significantly different during the corresponding time-periods post-contrast exposure (Tab. 3).

DISCUSSION

In accordance to our hypotheses, the incidence of AKI in the CE group was not higher than that in the NCE group. Exposure to radio-contrast twice within 24 hours also did not confer a higher CI-AKI risk than a single scan. One of the interesting findings of our study is the lower rate of AKI in contrast-exposed patients as compared with patients without any exposure to the nephrotoxic radio-contrast. This observation might be confounded by measures generally undertaken for CI-AKI prevention such as the liberal use of intravenous volume expansion in CE patients, possibly resulting in hemodilution and consequently lowering of sCr. However, this effect would not have persisted beyond weeks, and the 30-day delta-sCr for CE group was not inferior to that of NCE patients. Hospital-acquired AKI is often multi-factorial, and strongly associated with the magnitude of acute illnesses including sepsis and cardiovascular diseases, and drug-related renal injury (Kane-Gill *et al.*, 2015; Zeng *et al.*, 2014). These factors may exert greater influence on AKI, more so than contrast-exposure alone, hence explaining the AKI risk even in NCE patients. Patients with pre-existing CKD had higher risk of AKI (Mehran *et al.*, 2004). Caution should perhaps be focused on this sub-group of patients at risk.

Contrast-induced acute kidney injury

CI-AKI is the third most common cause of acute renal dysfunction, accounting for over 10% of hospital-associated AKI (Nash *et al.*, 2002). Various definitions of CI-AKI used include relative increase >50% over baseline sCr, or absolute increase in sCr of >0.5 mg/dL above baseline within days of



CT – computed tomography imaging.

Fig. 1. Flow diagram of the study

	NCE group (*n* = 93)	CE1 group (*n* = 203)	CE2 group (*n* = 169)	Entire cohort (*n* = 465)	*p* value
Age, years (range)	67 (18–92)	62 (25–92)	69 (32–92)	65 (18–92)	0.710
Diabetes mellitus (%)	34 (36.6)	64 (31.5)	56 (33.1)	154 (33.1)	0.213
Hypertension (%)	71 (76.3)	134 (66)	125 (74)	330 (71)	0.077
Baseline median creatinine, µmol/L (range)	92 (40–373)	78 (31–744)	88 (36–296)	85 (31–744)	0.081
Patients with baseline CKD, *n* (%)	12 (12.9)	17 (8.3)	14 (8.2)	44 (9.4)	0.082
CI-AKI, *n* (%)	9 (9.7)	5 (2.5)	4 (2.4)	18 (3.9)	0.004
Extended renal dysfunction, *n* (%)	6 (6.5)	3 (1.5)	1 (0.6)	10 (2.2)	0.185

AKI – acute kidney injury; **CE1** – contrast-exposed with 1 CTA scan; **CE2** – contrast-exposed with 2 serial CTA scans; **CKD** – chronic kidney disease; **CTA** – computed tomography angiography; **eGFR** – estimated glomerular filtration rate; **NCE** – no contrast-exposure scan.

Tab. 1. Baseline characteristics of the cohort

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AKI	**Odds ratio**	**95% CI**	***p* value**
Diabetes mellitus (Yes : No)	2.63	(1.02–6.81)	0.038
eGFR (per mL/min/1.73 m^2 decline)	1.02	(1.00–1.03)	0.048
Contrast-administration (Yes : No)	0.25	(0.10–0.65)	0.004
Extended renal dysfunction	**Odds ratio**	**95% CI**	***p* value**
Diabetes mellitus (Yes : No)	0.268	0.57–7.36	0.268
eGFR (per mL/min/1.73 m^2 decline)	1.006	1.00–1.01	0.007
Contrast-administration (Yes : No)	0.08	(0.01–0.81)	0.032
AKI – acute kidney injury; **CI** – confidence interval; **eGFR** – estimated glomerular filtration rate.			

Tab. 2. Multivariate analysis for prediction of AKI and extended renal dysfunction

	Early (range)	***p* value**	**Late (range)**	***p* value**
NCE group (reference)	−11.5 mmol/L (−59 to 285)	NA	−13 (−65 to 351)	NA
CE1 group	−11 mmol/L (−75 to 25)	0.583	−1.5 (−44 to 151)	0.417
CE2 group	−12 mmol/L (−50 to 8)	0.231	−10 (−36 to 48)	0.946
NCE – no contrast-exposure scan; **CE1** – contrast-exposed with 1 CTA scan; **CE2** – contrast-exposed with 2 CTA scans within 24 hours.				

Tab. 3. Median change in creatinine levels in the first 5 days (early) and after 30 days (late)

contrast exposure [Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012]. The use of eGFR has been shown to be a better indicator of renal function than sCr, and some radiologists use this measure to assess risk for CI-AKI (Benko *et al.*, 2007). A combination of factors is postulated to cause CI-AKI. Renal vasoconstriction and direct tubular injury are thought to be the main culprits. Contrast affects the renal vasculature by direct endothelial injury and dysfunction, leading to vasa recta constriction and medullary hypoperfusion and hypoxia (Sendeski *et al.*, 2012). Hypoxia-sensitive medullary thick ascending limb of the loop of Henle may be particularly susceptible to contrast-injury (Rosenberger *et al.*, 2005). Other mechanisms such as tubular acidosis, ischaemic injury from reactive oxygen species, and renal hypoxia from reduced cardiac output contribute to CI-AKI (Kurnik *et al.*, 1998; Margulies *et al.*, 1990; Persson *et al.*, 2005). Pre-existing CKD and the contrast load administered are associated with the incidence and prognosis of CI-AKI. Therefore, a baseline sCr determination has become a prerequisite for contrast-enhanced studies in many radiology practices.

Our findings are in agreement with the previous reports on CI-AKI in AIS patients (Hopyan *et al.*, 2008; Josephson *et al.*, 2005; Krol *et al.*, 2007). These studies reported the incidence of AKI as 3–6%. The incidence of dialysis related to contrast-enhanced imaging is also extremely low at <1% (Hopyan *et al.*, 2008; Josephson *et al.*, 2005; Krol *et al.*, 2007). Our finding of a low risk of CI-AKI (2.4%) is reassuring for the treating physicians (Oleinik *et al.*, 2009). We believe that the reductions in sCr during the first few days might have occurred due to the aggressive intravascular volume expansion that all AIS patients receive. Although, 10 of the 18 patients with CI-AKI developed extended renal dysfunction, none of them required dialysis. The low incidence of CI-AKI in these patients, even lower than AKI rate in the NCE group, suggests that the benefits of CTA possibly outweigh the purported renal risk in an acute stroke setting, without knowledge of admission sCr prior to CTA. The safety of using intravenous contrast agents in patients is enhanced with the use of contrast agents with lower osmolarity, and adequate intravenous circulatory volume expansion [Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012].

STRENGTHS AND LIMITATIONS

To the best of knowledge, our study is the largest case series on AKI risk with CTA in patients with AIS. Although this relationship is highly relevant in clinical practice, it remains under-reported in medical literature. We provide supportive evidence that the purported AKI risk in this scenario is low, and question the paradigm that patients with acute stroke be denied timely CTA and thrombolysis while awaiting an admission sCr. This is further strengthened by the observation that none of our patients needed acute dialysis and very few patients demonstrated persistent renal dysfunction beyond 30 days. However, certain limitations of our study need to be acknowledged. First, the retrospective nature of our study might be subjected to bias with regards to patient selection for CTA, with patients of higher AKI risk being offered non-contrasted imaging. Second, our study was also notably limited by the fact that AKI rates were very low and hence minute differences in outcome may not be statistically significant. However, our

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large patient cohort allows a robust estimation of risk, and such low event rates may not be clinically significant anyway, with regards to emergent decision-making for imaging modality. Finally, we did not differentiate CI-AKI from AKI due to other causes, and many factors such as critical illness, hemodynamic shock and nephrotoxins might have contributed to renal dysfunction, but this is a common limitation across most studies on this subject. Moreover, fluctuations in sCr are not uncommon in hospitalized patients, with or without contrast exposure (Newhouse *et al.*, 2008).

CONCLUSION

Emergent or serial CTAs in AIS were associated with very low risk of AKI and extended renal dysfunction at 30 days, and these risks were not significantly higher than in AIS patients without early contrast exposure. Our findings suggest that we may not have to delay vital neurological imaging for purpose of pre-scan renal function assessment, due to the critical therapeutic window for urgent thrombolysis, and overall low risk of renal dysfunction. Patients with diabetes mellitus or CKD should be counselled on the low CI-AKI risk, but this should be assessed along with the potential of enhanced neurological and functional prognosis with emergent CTA and thrombolysis.

Conflict of interest

None of the authors declare any conflicts of interest or competing interests.

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