Research in Diagnostic and Interventional Imaging 7 (2023) 100032

Contents lists available at ScienceDirect

Research in Diagnostic and Interventional Imaging

journal homepage: https://www.journals.elsevier.com/redii

Original article

Changes in blood and renal function in patients after cerebral digital subtraction angiography

Juan Manuel Marquez-Romero[a,*], Marco Zenteno[b], Antonio Arauz[c]

[a] Neurology. HGZ #2, IMSS, Aguascalientes Oriente SN, Aguascalientes 20190, Mexico
[b] Department of Neurological Endovascular Therapy, Instituto Nacional de Neurología y Neurocirugía "MVS, Mexico City, Mexico
[c] Stroke Clinic, Instituto Nacional de Neurología y Neurocirugía "MVS", Mexico City, Mexico

ARTICLE INFO

Article History:
Received 15 March 2023
Accepted 9 August 2023
Available online 1 September 2023

Keywords:
Contrast induced nephropathy
Digital substraction angiography
Cerebrovascular disease

ABSTRACT

Objective: Describe the incidence of contrast-induced acute renal injury (CI-AKI) and the changes in hematocrit in a cohort of patients undergoing elective cerebral digital subtraction angiography (DSA).
Methods: In this prospective study, patients undergoing cerebral DSA were assessed for hematocrit level and CI-AKI risk factors before the procedure and for developing CI-AKI 72 h after exposure to the contrast media.
Results: Among 215 patients (109 men, mean age 36.6 years). The most frequently found CI-AKI risk factor was hypertension. There were no cases of permanent renal impairment after 14 days. Significant changes were observed in hematocrit (45.7 ± 4.9, vs. 44.5 ± 4.6, p = 0.001), estimated creatinine clearance (129.7 ± 48.3, vs. 123.1 ± 40.5, p = 0.002), and serum creatinine (0.72 ± 0.19, vs 0.74 ± 0.18, p = 0.031). The mean change in serum creatinine 72 h after contrast administration was +0.27 ± 0.10 mg/dL ($p < 0.05$).
Conclusions: The incidence of CI-AKI after elective cerebral DSA was 1.4%. A significant decrease in hematocrit was observed up to 72 h after the procedure.

1. Introduction

Cerebrovascular disease is increasingly managed with endovascular techniques, and the growing number of patients has highlighted the importance of previously neglected situations [1]. One such issue is the need to perform cerebral digital subtraction angiography (cerebral DSA) as a follow-up study. Although the incidence of complications in patients undergoing elective cerebral DSA is very low, risks such as cerebral thromboembolism, Contrast-induced acute kidney injury (CI-AKI), allergic reactions, and scarring of the groin occur in about 0.34−1.3% of the patients, and, the risk accumulates with repeating studies [2]. In addition to cerebral DSA, other imaging modalities may share some of the risks mentioned above, especially the risk for CI-AKI. Depending on the series consulted, the definition used, and the population studied, the reported incidence of CI-AKI ranges from 2% in acute stroke patients [3] to 22.8% in neurosurgical patients [4]. In addition to the risk of CI-AKI, contrast administration has been related to both hemodynamic and rheological effects [5], such as osmotic shrinkage of red blood cells leading to an osmolality-dependent decrease in hematocrit both in normal and sickle cell blood donors [6]. Data from animal models shows that exposure to hyperosmotic contrast medium induces in addition to cell shrinkage, a decrease in the water permeability of the cellular plasma membranes, thus compromising the ability to regulate cellular volume ultimately resulting in hemolysis [7]. In human blood cells, added to cell shrinking, cellular swelling is also present. Still, the high osmolality of contrast media does not explain the degree of shrinkage and subsequent swelling. It has been theorized that physicochemical properties of the contrast media must be involved [8].

Hitherto, low hematocrit has been recognized as a risk factor for the development of CI-AKI, but the incidence of low hematocrit after contrast administration has not been reported previously [9].

Thus, this prospective study aimed to describe the incidence of CI-AKI and the changes observed in the hematocrit of a cohort of patients undergoing elective cerebral DSA.

2. Materials and methods

2.1. Ethics statement

The institutional review board of the Instituto Nacional de Neurología y Neurocirugía "MVS," Mexico City, Mexico, approved the protocol and informed consent form. All patients provided signed informed consent before participating.

* Corresponding author.
E-mail address: scint1st@gmail.com (J.M. Marquez-Romero).

https://doi.org/10.1016/j.redii.2023.100032

2.2. Participants

This is a cohort study of all consecutive patients scheduled for cerebral DSA. To be included in this report, the patients had to be older than 18 (with no upper age limit) and have a complete blood count and serum creatinine concentration measured within seven days before the procedure.

Criteria for exclusion were pregnancy, lactation, intravascular administration of an iodinated contrast medium within the previous seven days, treatment with metformin or NSAIDs within the last 48 h, intake of nephrotoxic drugs within the previous seven days, history of allergic reactions to iodinated contrast media, newly discovered unstable diabetes, renal transplantation, or end-stage renal disease necessitating dialysis.

2.3. Data collection

The data registered included age, sex, coagulation parameters, number and type of concomitant medications, specifically antihypertensive, antiepileptics, and antiplatelet agents, indication for cerebral DSA, the volume of iodinated contrast media used, serum creatinine concentration, and hematocrit level. Renal function was classified according to the stages set by the National Kidney Foundation, with creatinine clearances ≥ 90 mL/min considered normal, 60 to 89 mL/min considered mildly impaired, 30 to 59 mL/min considered moderately impaired, and ≤ 30 mL/min considered severely impaired [10]. The CI-AKI risk score was calculated as specified by Mehran et al. [11].

Briefly, the score rates the presence of risk factors for CI-AKI (Congestive heart failure, Hypotension, use of intraaortic balloon pump, age > 75 years, anemia, diabetes mellitus, administered contrast volume and either estimated glomerular filtration rate (mL/min 1.73 m^2) or serum creatinine > 1.5 mg/dL) in a scale from one to 25 points. Risk categories for the development of CI-AKI include low (≤ 5 points), moderate (6−10 points), high (11−15 points), and very high (≥ 16 points).

2.4. Procedures

All patients received standard intravenous hydration with isotonic saline (0.9% sodium chloride, 1 mL/kg/hour for 4 h before and 8 h after the procedure). Following angiography, the patients were monitored for 8 h in a recovery room and then discharged. After release from the hospital, a subsequent serum creatinine and hematocrit measurement was scheduled 72 h after cerebral DSA to maximize the detection of patients following the proposed epidemiology of CI-AKI [12]. All measurements were performed in the same laboratory. CI-AKI was defined following the Kidney Disease Global Outcomes (KDIGO) guidelines as an increase in serum creatinine of 0.3 mg/dL or greater within 48 h of contrast use or a 50% or greater increase from baseline serum creatinine within seven days [13].

2.5. Statistical analysis

The data are presented in frequency tables. The Kolmogorov-Smirnov test was used to verify the normality of the data distribution. According to the observed distribution, numerical variables are described with either a mean (SD) or a median (range). Categorical variables are expressed as percentages. A paired-sample *t*-test was used to compare changes in continuous variables pre- and postcerebral DSA. incidence was calculated from contingency tables and the 95% confidence interval was calculated with Poisson regression model. The alpha level was set at 0.05. All the analyses were calculated using the Statistical Package for the Social Sciences ™ version 17.0 for Windows ™.

3. Results

3.1. Demographic data

During the recruitment period, 297 patients underwent elective cerebral DSA and were thus eligible for participation in this study. Among them, 12 patients (4.0%) met the exclusion criteria (nine patients underwent CTA (3.0%), two were pediatric patients (0.7%), and one patient (0.3%) reported a previous non-DSA-related severe allergic reaction to iodinated contrast media). Precerebral DSA data was recollected from 280 patients (94.3%). However, 40 patients (13.5%) were excluded from the analysis due to a lack of follow-up laboratory test results. Therefore, data from 215 patients were analyzed in this report.

The mean age of the patients was 36.61 ± 15.4 years, 109 male (50.7%). Indications for cerebral DSA included diagnostic evaluation of arteriovenous malformations (AVM), 57%, follow-up of treated intracranial aneurysm (26%), dural arteriovenous fistula (4%), and carotid atherosclerosis (3%). Other disorders accounted for the remaining 10% and included: follow-up of cervical artery dissection and diagnostic evaluation of cerebral venous thrombosis or intracranial tumors.

3.2. Risk factors for CI-AKI

Basal population characteristics are outlined in Table 1. All patients had normal renal function. The most frequently encountered comorbidity was high blood pressure, which was present in 27 patients (12.6%), followed by diabetes in six patients (2.8%), and there

Table 1
Basal characteristics and indications for cerebral angiography in 215 patients undergoing cerebral digital substraction angiography.

Female, n (%)	106 (49.3)
Age (years)	36.61 ± 15.4
Diabetes mellitus, *n* (%)	6 (2.8)
Hypertension, *n* (%)	27 (12.6)
Cardiac failure, *n* (%)	0 (0)
Medication use, *n* (%)	32 (15.0)
Antihypertensives	27 (12.6)
Telmisartan	6 (2.8)
Captopril	2 (1.0)
Amlodipine	1 (0.5)
Metoprolol	1 (0.5)
Hydralazine	1 (0.5)
Antidiabetics	
Glibenclamide	6 (2.8)
Antiepileptics	
Phenytoin	4 (1.9)
Weight (kg)	68.3 ± 14.9
Height (cm)	161.9 ± 10.7
BMI (kg/m^2)	25.9 ± 4.4
Mehran Score	1.6 ± 1
Mild, n (%)	
- 1 point	128 (59.4)
- 2 points	72 (33.9)
- 3 points	0 (0)
- 4 points	9 (4.1)
- 5 points	3 (1.3)
Medium, n (%)	
- 7 points	0 (0)
- 8 points	3 (1.3)
Contrast medium volume (cm^3)	132.3 ± 49.1
Indication for angiography, *n* (%)	
Arteriovenous malformation	123 (57.2)
Follow-up of treated aneurysm	56 (26.0)
Follow-up of carotid stenting	6 (2.8)
Follow-up of dural arteriovenous fistula embolization	9 (4.2)
Other	22 (10)

Unless otherwise specified, the data are reported as the mean ± standard deviation.

Table 2
Comparison of pre and post angiography laboratory results for the 215 patients included in the analysis.

	Pre angiography	Post angiography	*p*
Hematocrit	45.7 ± 4.9	44.5 ± 4.6	0.001
Serum creatinine concentration (mg/dL)	0.72 ± 0.19	0.74 ± 0.18	0.031
Glomerular filtration rate (ml/min)	129.7 ± 48.3	123.1 ± 40.5	0.002

were no previous renal or cardiac failure cases. All patients with high blood pressure were receiving medication, four patients (1.9%) were taking antiepileptics, and one patient (0.5%) was taking both medication groups. No patient received antiplatelets. Therefore 32 patients (15.0%) were taking at least one medication. According to the Mehran risk score, all but three patients were categorized as low risk. The three patients in the moderate risk group were given a score of 8, and two of them developed CI-AKI. The mean volume of contrast administered was 132.3 ± 49.1 cm^3. In all cases, ioversol was used (Optiray™ 320). We detected modifications in laboratory parameters in the patients after contrast administration (Table 2). Significant changes were observed in hematocrit, estimated glomerular filtration rate, and serum creatinine. The mean change in serum creatinine 72 h after contrast administration was +0.27 ± 0.10 mg/dL ($p < 0.05$). Fig. 1 depicts the relation between the change in serum creatinine pre and post-cerebral DSA and the amount of contrast injected.

3.3. Incidence of CI-AKI and follow-up

Three patients developed CI-AKI; thus, this study's incidence was 1.4% (95% CI 0.2 − 3.0). All the patients had an elevation of creatinine above 0.3 mg/dL 72 h after the procedure (0.39, 0.57, and 0.98 mg/dL). The two patients with elevations higher than 0.5 mg/dL were referred to a nephrology department and monitored as outpatients. The patient with 0.39 mg/dL of creatinine elevation was observed as an outpatient in the Neuroendovascular clinic. The serum creatinine levels of these patients returned to normal within two weeks (8, 9, and 14 days after contrast administration). The calculated creatinine clearance was never below 60 mL/min in either patient, reflecting a mildly decreased glomerular filtration ratio according to the stages set by the National Kidney Foundation.

4. Discussion

CI-AKI is the manifestation of damage to the kidney induced by a contrast medium injection. The delayed onset time of this disorder precludes its detection in outpatients, which are usually rapidly discharged after the procedure. Thus, the problem can go unnoticed by the patient and the physician. CI-AKI has been widely studied in cardiological patients undergoing coronary angiography, where the prevalence of risk factors for CI-AKI is high, but information for neurological patients is scarce (Table 3). In a non-emergency setting, CI-AKI incidence in patients undergoing coronary angiography is 6.1−8.5% [14]; however, to our knowledge, the incidence of patients undergoing elective cerebral DSA has never been described.

CI-AKI is the third cause of acute kidney impairment in hospitalized patients undergoing coronary angiography. It may be present in 1−6% of unselected subjects and 40−50% of high-risk populations [15]. CI-AKI incidence also depends on the definition used and ranges from 10 to 38% depending on the extent of increase in serum creatinine levels required to fulfill the definition [16].

Regarding risk factors, cardiological patients, the widespread use of potentially nephrotoxic medications adds to the risk conferred by their high incidence of diabetes, non-controlled high blood pressure, and cardiac failure [17]. In the present study, the prevalence of risk factors for CI-AKI was low (12.6% of high blood pressure and 2.8% of diabetes), with only 1.3% of the patients (n = 3) being rated in the moderate risk category of the Mehran scale. Age is another risk factor, and coronary patients are generally older than 60 years. The mean age of our population was 36.6, which is more than two decades

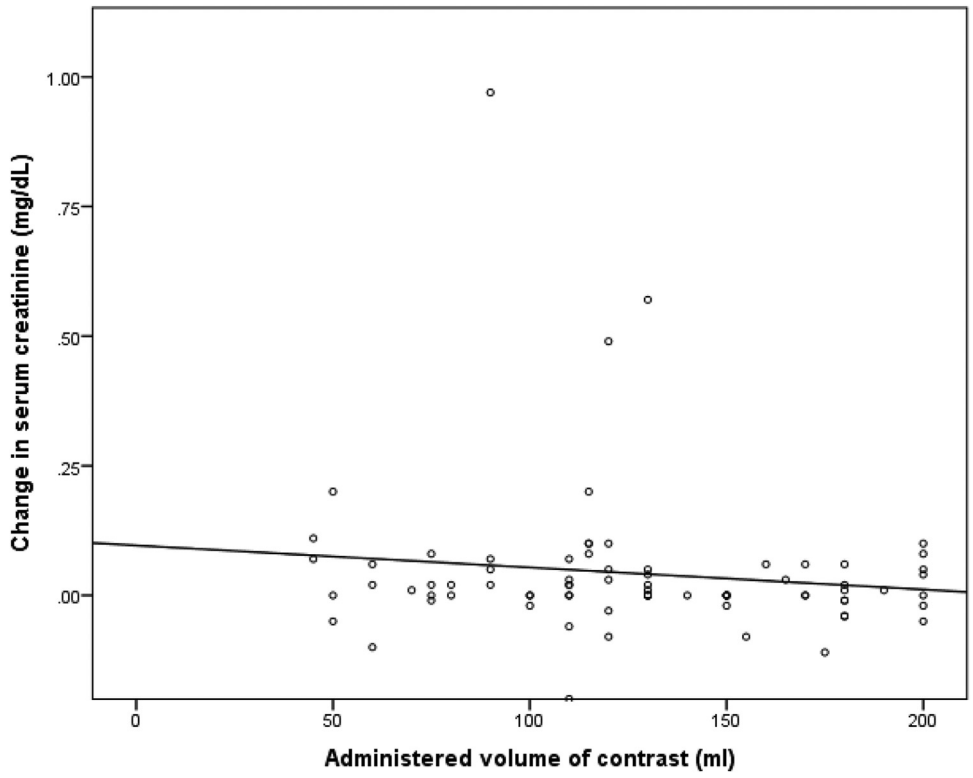

Fig. 1. Scatter plot of change in serum creatinine and volume of administered contrast in the sample (n = 215).

Table 3
Previously published studies addressing contrast-induced nephropathy in cerebrovascular disease patients.

Study	Population, setting	Number of patients	Contrast route of administration	Imaging modality	CI-AKI incidence	Prospective	CI-AKI risk factors studied
Josephson et al., 2005 [23]	Stroke patients, (ED)	1075	Intravenous	CTA, CT perfusion	4.8%	No	No
Krol et al., 2007 [27]	Stroke patients, (ED)	224	Intravenous	CTA	3.0%	No	No
Dittrich et al., 2007 [3]	Stroke patients, (ED)	162	Intravenous	CTA, CT perfusion	2.0%	No	Yes
Hopyan et al., 2008 [18]	Stroke patients, (ED)	198	Intravenous	CTA, CT perfusion	2.9%	No	No
Oleinik et al., 2009 [20]	Stroke patients, (Inpatients)	348	Intravenous	CTA	6.0%	No	Yes
Lima et al., 2010 [19]	Stroke patients, (inpatients)	575	Intravenous	CTA, CT perfusion, cerebral DSA	5.0%	No	Yes
Serafin et al., 2011 [4]	Neurosurgical patients, (inpatients)	92	Intra-arterial	cerebral DSA	22.8%	Yes	Yes
Present series, 2012	Cerebrovascular disease (outpatients)	215	Intra-arterial	cerebral DSA	1.4%	Yes	Yes

CI-AKI: contrast-induced nephropathy, ED: emergency department, DSA: digital subtraction angiography, CT: computed tomography, CTA: computed tomography angiography.

younger. The low prevalence of these risk factors explains the low incidence and good outcome of CI-AKI in the present study.

An additional consideration is that this report includes data from outpatients. Therefore, they differ from patients whose indication for cerebral DSA is acute cerebrovascular disorders. A study on neurosurgical patients to whom intra-arterial contrast was administered showed a CI-AKI prevalence of 22.8% [4], much higher than our findings. The apparent differences in the prevalence of CI-AKI risk factors among acute neurosurgical and elective patients explain this difference. Despite a slightly higher mean age of patients (49.6 ± 12.6 vs. 36.6 ± 15.4 years), the volume of contrast medium (151.2 ± 52.1 cm^3 vs. 132.3 ± 49.1 cm^3) and the estimated glomerular filtration rate (97.8 ± 26.3 mL/min/1.73 m^2 vs. 143.1 ± 51.1 mL/min/1.73 m^2) were similar in both populations. However, the prevalence of high blood pressure in acute patients differed by twofold (33.7% vs. 12.6%). Further, 38% of the acute patients were taking NSAIDs, 18% were using ACE inhibitors, and 3.3% had cardiac failure. These data suggest that the prevalence of CI-AKI risk factors is higher in acute neurosurgical patients.

Another comparable population was assessed by Hopyan et al. [18]. This study consisted of 198 patients admitted for acute stroke that underwent imaging with CT angiography and perfusion CT, yielding a 2.9% prevalence of CI-AKI. A total of 11% of these patients with CI-AKI were diabetic. This population did not undergo cerebral catheterization, but they are patients with an acute neurological disorder receiving contrast imaging. In a study by Lima et al. [19], 38 out of 575 patients received cerebral DSA and CTA. Here, the authors report a lower incidence of CI-AKI in contrast-exposed patients than those of a non-exposed group (5% vs. 10%); however, the proportion of patients with diabetes and congestive heart failure was significantly higher in the non-contrast group. Patients in the control group were also older and had higher baseline creatinine levels. The finding of a lower incidence of CI-AKI in contrast-exposed patients was also described by Oleinik et al. [20] in patients with intracerebral hemorrhage. Again, CI-AKI risk factors were higher in the non-exposed group.

The prevalence of CI-AKI in acute stroke patients is nearly three times higher than in our population, and the prevalence of risk factors is also higher. A thorough analysis of the acute stroke population may find subtle differences because patients with an atherosclerotic stroke might be more susceptible to CI-AKI than those with cardiac embolic stroke or other non-atherosclerotic pathologies; this is because an increased renal resistive index is associated with the severity of systemic atherosclerosis [21], and a high renal resistive index is a risk factor for CI-AKI [22]. In our study, patients with atherosclerosis are underrepresented, as AVM was the main indication for cerebral DSA.

In a retrospective study of 1075 patients receiving stroke protocol CT scans that included the IV administration of 150 cm^3 of contrast agent, 52 patients (4.8%) experienced increases in the serum creatinine level of ≥0.5 mg/dl, but the medical team considered contrast nephropathy as the etiology of this increase in creatinine in only four of the 1075 patients (0.37%) [23]. In two other studies with similar methodology, the incidence of CI-AKI was 2 and 3% [3,23]; however, the risk factors were not adequately described, and only patients who received a stroke protocol CT were studied.

In addition to CI-AKI, we found statistically significant changes from baseline to 72 h creatinine, creatinine clearance, and hematocrit. Although insufficient to fulfill the definition of CI-AKI, the changes in serum markers of kidney function demonstrate a deleterious effect on kidney function with contrast administration. Furthermore, the decrease in hematocrit can modify the flow and coagulation properties of the patient's blood. Such changes have been reported immediately after diagnostic cardiac catheterization [24]. The results of the present study show that these changes persist up to 72 h after the procedure. The clinical significance of the data above is relevant when aggravating risk factors are present or if there is a need for repeating studies. Other higher-risk populations include patients with chronic anemia (especially sickle cell disease) [6] and patients scheduled for major surgery and high bleed-risk procedures [25].

We acknowledge that several caveats can be ascribed from the findings of our study. First, the prevalence of CI-AKI is low due to recruitment bias because our population consists only of outpatients, and hospitalized patients with more serious neurological conditions were not evaluated. Second, 13.5% of eligible patients had to be excluded from the analysis due to a lack of follow-up laboratory results. The completion of these tests is difficult in our population because most patients are reluctant to incur additional financial charges after the procedure; many of them come from rural areas outside Mexico City, making it very difficult for them to return to the hospital unless there is an urgent need to do so (i.e., discomfort, pain). Finally, our hospital constitutes a national reference center for AVM treatment, which explains why this was the most frequent indication for cerebral DSA. The characteristics of patients with AVM closely resemble those of the patients in this study (i.e., young age, very low prevalence of chronic diseases) [26]. Additionally, our center is a teaching hospital, and most of the cerebral DSA were performed by operators under training which led to an increased number of injections thus augmenting the amount of contrast media administered. These characteristics of our institution explain the volume of patients scheduled for follow-up cerebral DSA, usually performed between one and three months after the therapeutic procedure (embolization of aneurysm or dural arteriovenous fistula and carotid stenting). Still, for AVM, the patients can undergo multiple

procedures, for example, diagnostic cerebral DSA, angiography-based stereotactic radiosurgery, adjuvant embolization follow-up, postsurgical cerebral DSA, etc. The high number of AVM-related indications for cerebral DSA undoubtedly skewed the sample in this study.

5. Conclusion

In conclusion, CI-AKI incidence in cerebrovascular disease patients undergoing elective cerebral DSA is very low. The low prevalence of CI-AKI risk factors and the younger age of the patients explains the low incidence. cerebral DSA is a safe and well-tolerated procedure from the renal point of view. In addition, contrast media administration leads in vivo to a significant decrease in hematocrit, which is still present 72 h after administration. The clinical significance of this change is uncertain. However, it may be relevant in anemic patients or those undergoing surgery.

Funding

None.

Ethics statement

The institutional review board of the Instituto Nacional de Neurología y Neurocirugía "MVS," Mexico City, Mexico, approved the protocol and informed consent form. All patients provided signed informed consent before participating.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Juan Manuel Marquez-Romero: Conceptualization, Writing – review & editing, Writing – original draft, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization. **Marco Zenteno:** Conceptualization, Writing – review & editing, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation. **Antonio Arauz:** Writing – review & editing, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation.

Acknowledgments

The authors are indebted to the nursing staff from the outpatient cerebral angiography unit.

References

[1] Al-Ameri H, Thomas ML, Yoon A, Mayeda GS, Burstein S, Kloner RA, et al. Complication rate of diagnostic carotid angiography performed by interventional cardiologists. Catheter Cardiovasc Interv 2009;73(5):661–5.
[2] Dawkins AA, Evans AL, Wattam J, Romanowski CA, Connolly DJ, Hodgson TJ, et al. Complications of cerebral angiography: a prospective analysis of 2,924 consecutive procedures. Neuroradiology 2007;49(9):753–9.
[3] Dittrich R, Akdeniz S, Kloska SP, Fischer T, Ritter MA, Seidensticker P, et al. Low rate of contrast-induced nephropathy after CT perfusion and CT angiography in acute stroke patients. J Neurol 2007;254(11):1491–7.
[4] Serafin Z, Karolkiewicz M, Gruszka M, Strozecki P, Lasek W, Odrowaz-Sypniewska G, et al. High incidence of nephropathy in neurosurgical patients after intra-arterial administration of low-osmolar and iso-osmolar contrast media. Acta Radiol 2011;52(4):422–9.
[5] Pugh ND. Haemodynamic and rheological effects of contrast media: the role of viscosity and osmolality. Eur Radiol 1996;6(2):S13–5.
[6] Losco P, Nash G, Stone P, Ventre J. Comparison of the effects of radiographic contrast media on dehydration and filterability of red blood cells from donors homozygous for hemoglobin A or hemoglobin S. Am J Hematol 2001;68(3):149–58.
[7] Galtung HK, Loken M, Sakariassen KS. Effect of radiologic contrast media on cell volume regulation in rabbit proximal renal tubules. Acad Radiol 2001;8(5):398–404.
[8] Galtung HK, Sorlundsengen V, Sakariassen KS, Benestad HB. Effect of radiologic contrast media on cell volume regulatory mechanisms in human red blood cells. Acad Radiol 2002;9(8):878–85.
[9] Myung JW, Kim JH, Cho J, Park I, Kim HY, Beom JH. Contrast-induced acute kidney injury in radiologic management of acute ischemic stroke in the emergency setting. AJNR Am J Neuroradiol 2020;41(4):632–6.
[10] National Kidney F. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 2002;39(2):S1–266.
[11] Mehran R, Aymong ED, Nikolsky E, Lasic Z, Iakovou I, Fahy M, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. J Am Coll Cardiol 2004;44(7):1393–9.
[12] McCullough PA. Contrast-induced nephropathy: definitions, epidemiology, and implications. Interv Cardiol Clin 2014;3(3):357–62.
[13] Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pract 2012;120(4):c179–84.
[14] Weisbord SD, Hartwig KC, Sonel AF, Fine MJ, Palevsky P. The incidence of clinically significant contrast-induced nephropathy following non-emergent coronary angiography. Catheter Cardiovasc Interv 2008;71(7):879–85.
[15] Waybill MM, Waybill PN. Contrast media-induced nephrotoxicity: identification of patients at risk and algorithms for prevention. J Vasc Interv Radiol 2001;12(1):3–9.
[16] Lautin EM, Freeman NJ, Schoenfeld AH, Bakal CW, Haramati N, Friedman AC, et al. Radiocontrast-associated renal dysfunction: incidence and risk factors. AJR Am J Roentgenol 1991;157(1):49–58.
[17] Yu HT, Kim KJ, Bang WD, Oh CM, Jang JY, Cho SS, et al. Gender-based differences in the management and prognosis of acute coronary syndrome in Korea. Yonsei Med J 2011;52(4):562–8.
[18] Hopyan JJ, Gladstone DJ, Mallia G, Schiff J, Fox AJ, Symons SP, et al. Renal safety of CT angiography and perfusion imaging in the emergency evaluation of acute stroke. AJNR Am J Neuroradiol 2008;29(10):1826–30.
[19] Lima FO, Lev MH, Levy RA, Silva GS, Ebril M, de Camargo EC, et al. Functional contrast-enhanced CT for evaluation of acute ischemic stroke does not increase the risk of contrast-induced nephropathy. AJNR Am J Neuroradiol 2010;31(5):817–21.
[20] Oleinik A, Romero JM, Schwab K, Lev MH, Jhawar N, Delgado Almandoz JE, et al. CT angiography for intracerebral hemorrhage does not increase risk of acute nephropathy. Stroke 2009;40(7):2393–7.
[21] Ohta Y, Fujii K, Arima H, Matsumura K, Tsuchihashi T, Tokumoto M, et al. Increased renal resistive index in atherosclerosis and diabetic nephropathy assessed by Doppler sonography. J Hypertens 2005;23(10):1905–11.
[22] Perrotta AM, Gigante A, Rotondi S, Mene P, Notturni A, Schiavetto S, et al. Contrast-induced acute kidney injury and endothelial dysfunction: the role of vascular and biochemical parameters. J Pers Med 2023;13(4):701.
[23] Josephson SA, Dillon WP, Smith WS. Incidence of contrast nephropathy from cerebral CT angiography and CT perfusion imaging. Neurology 2005;64(10):1805–6.
[24] Scheller B, Hennen B, Thunenkotter T, Mrowietz C, Markwirth T, Schieffer H, et al. Effect of X-ray contrast media on blood flow properties after coronary angiography. Thromb Res 1999;96(4):253–60.
[25] Aspelin P, Nilsson PE, Schmid-Schonbein H, Schroder S, Simon R. Effect of four non-ionic contrast media on red blood cells in vitro. II. Aggregation. Acta Radiol Suppl 1987;370:85–7.
[26] Hernesniemi JA, Dashti R, Juvela S, Vaart K, Niemela M, Laakso A. Natural history of brain arteriovenous malformations: a long-term follow-up study of risk of hemorrhage in 238 patients. Neurosurgery 2008;63(5):823–9 discussion 9-31.
[27] Krol AL, Dzialowski I, Roy J, Puetz V, Subramaniam S, Coutts SB, et al. Incidence of radiocontrast nephropathy in patients undergoing acute stroke computed tomography angiography. Stroke 2007;38(8):2364–6.