Original Paper

Lack of Serum Creatinine Decrease After Coronary Angiography Despite Prophylactic Hydration After Routine Coronary Angiography/Angioplasty in Stable Angina Patients - Pilot Study

Pawel Burchardt^{a,b} Janusz Rzezniczak^b Tomasz Synowiec^c Dariusz Angerer^b
Anna Palasz^d Jakub Zurawski^a

^aDepartment of Biology and Environmental Sciences, ^bDepartment of Cardiology, J. Strus Hospital,

^cDepartment of General and Vascular Surgery and Angiology, ^dDepartment of Cardiology-Intensive Therapy, Poznan University of Medical Sciences, Poznan, Poland

Key Words

Contrast induced acute kidney injury • Coronary angiography • Coronary angioplasty • Iodine contrast

Abstract

Background/Aims: To prevent contrast induced renal dysfunction a periprocedural prophylactic hydration is applied. Due to dilution it should cause a drop in serum creatinine concentration (SCR). Surprisingly, no reduction in SCR after contrast admission is found in up to 25% of patients as early as 12-18 hours after coronary angiography/angioplasty. This study aims to find a clinical explanation as well as predict circumstances for this phenomenon.

Methods: Retrospective clinical and laboratory data was used from 341 patients who underwent elective coronary angiography/angioplasty, received a prophylactic hydration, and had serum creatinine concentration measured prior to, and 12-18 hours after invasive procedure with iodine contrast administration. To exclude an improper hydration due to no creatinine decrease, the number of red blood cells was analysed as well as hemoglobin and hematocrit in blood donations collected during the study time points. **Results:** The resulting lack of serum creatinine reduction could be explained by dehydration (measured by increase in number of RBC, HGB and HCT) only in 13.5%, 10.8%, and 20% of cases, respectively. Any form of abnormal glucose metabolism combined with either baseline serum creatinine concentration <0.87 mg/dL or creatinine clearance >86.77 mL/min, or GFR by CKD EPI >80.08 mL/min/1.73 m², or GFR by MDRD >74.48 mL/min/1.73 m² were the predictors for no creatinine decrease at outcome. Additionally, it was demonstrated that the lack of creatinine

decrease was more often observed among those patients whose initial renal function was better than in the subjects with reduction of SCR. **Conclusions:** This observation requires further prospective investigation on extended group of patients.

© 2016 The Author(s)
Published by S. Karger AG, Basel

Introduction

It is reasonable to screen patients to determine the group of people at high risk of contrast induced acute kidney injury (CI-AKI). Epidemiological studies showed that heart failure (HF), diabetes mellitus (DM) or usage of angiotensin convertase enzyme inhibitors (ACE-i), are considered to be the risk factors for CI-AKI [1-6]. Additionally Ribiccini et al found that after 12 h after angiography 5 to 10% creatinine shift had high sensitivity as well as specificity in classical contrast induced AKI recognition [7]. In our previous work it was described that up to 25% of patients showed no reduction in serum creatinine concentration as early as 12-18 hours after elective angiography/angioplasty with a concomitant prophylactic hydration [8]. Following this rationale, the present study examines (on the basis of hemoglobin, red blood cell /RBC/ count and creatinine concentrations /SCR/) whether SCR reduction in control tests after 12-18 h post coronary angiography/coronary angioplasty is, despite intense hydration, an early exponent of contrast-induced impairment of renal excretory function or just a cause of improper hydration.

It remains unresolved whether this phenomenon can be predicted on the basis of the assessment of initial renal function or any available laboratory parameters routinely used in clinical practice. In other words, if there are threshold values of parameters which may predict lack of early creatinine decrease. Thus, the second aim of the current study is to identify and characterize baseline renal function parameters and to possibly define their cut-off values at which the risk of lack of early creatinine decrease is clinically significant for the studied group. Additionally, we compare which of the routinely used parameters describing renal function could be the most effective in predicting the lack of early creatinine decrease.

Materials and Methods

Patients and clinical evaluation

Protocol of the study was approved by the local bioethics committee. All of the patients were informed about the procedure and signed the consent forms.

Analyses were carried out with data from consecutive 341 subjects admitted for elective, routine coronary angiography. Subjects with acute coronary syndrome or who had acute coronary syndrome less than 5 weeks prior to the study, were excluded, as well as people suffering from heart failure according to NYHA III-IV and also those with reduced ejection fraction, i.e. below 30%. Various clinical data concerning patient's health were collected from existing medical records and clinical examinations.

The characteristics of the subjects is shown in Table 1. Patients were hydrated intravenously with saline enriched with 0.038 g/100 mL of KCl, 0.0394 g/100 mL of (CaCl₂·6H₂O), 0.02 g/100 mL, (MgCl₂·6H₂O), 0.462 g/100 mL (CH₃COONa·3H₂O), 0.09 g/100 mL (C₆H₅Na₃O₇·2H₂O) (Fresenius Kabi, Poland) for a total of 24 hours, including the time before, during and after coronary angiography/angioplasty [9]. All of the patients finished intravenous periprocedural irrigation at the same time, but the angiographies were performed at different points in time of the hydration procedure. No reduction in serum creatinine was define as any SCR increase in control test (included 0).

Two types of low osmolar contrast agents were used for angiographies: IOMERON 350 (Bracco Imaging, Germany) and Optiray 350 (Tyco Healthcare, Germany).

Biochemical analyses

Serum creatinine concentration was determined according to Jaffe's reaction [10] using Roche Cobas C (Hitachi, Germany) /non IDMS method/. Creatinine clearance was fixed according to Cockcroft-Gault

[11, 12] formula. Glomerular filtration rate was estimated using formulas of, CKD EPI (Chronic Kidney Disease Epidemiology Collaboration) [13] and MDRD (Modification of Diet in Renal Disease), respectively [14]. Assessments of peripheral blood counts were performed using (Sysmex XT2000i, USA) system. Concentration of urea, uric acid, total cholesterol, triglycerides and high-density lipoproteins were quantified using enzymatic colorimetric method (Cobas C Roche/Hitachi, Germany) with specific reagents. The concentration of low-density lipoproteins was calculated using the Friedewald formula. Potassium and sodium concentrations were determined by potentiometry using Cobas System 6000 (Roche Diagnostics, Germany).

Statistical analyses

The quantitative data is presented as mean $\pm$ standard deviation (SD). The distribution of continuous data was analysed with the Shapiro-Wilk test. Continuous data were compared by the t-test or Mann-Whitney U test. For parameters that showed significant difference, the receiver-operator curve (ROC) were used. For parameters with the area under the curve (AUC) statistically different from 0.5, proper cut-off thresholds for lack of creatinine decrease in control tests were established with the 'classification tree' method.

For data with binomial distribution, binomial proportion test was applied to compare the data between patients with and without early creatinine decrease at outcome.

Parameters related to lack of creatinine decrease were put to univariate and multivariate logistic regressions. Statistical significance was set at $p < 0.05$. All analyses were performed with Statistica 7.0 (Statsoft, USA) and SPSS-20 (IBM, USA).

Results

Characteristics

The study group consisted of 341 patients aged 60.9 ± 8.6 years, 61% were men, average BMI in the study population was 29.1 ± 5 [kg/m²]. The following numbers of patients suffered from: heart failure 60 (16.8%) NYHA I-II with EF between 50-31%, chronic kidney disease 35 (9.8%), hypertension 292 (81.2%), disturbances of carbohydrate metabolism (glucose intolerance, impaired fasting glucose and diabetes combined) 153 (42.9%). Previous history

Table 1. Characteristics of studied group

	Studied group Mean $\pm$ SD (N=341)
Age [years]	60.9 $\pm$ 8.5
BMI kg/m ²	29.1 $\pm$ 4.02
Hypertension %	85.6
Glucose metabolism disturbances (IFG, IGT, DM) %	44.9
Heart failure %	17.6
Previous myocardial infarction %	37
Previous PTCA %	56.3
Dyslipidemia %	37
Chronic kidney disease [%] (GFR<60 [mL/min/1.73m ²])	10.3
Creatinine baseline [mg/dL]	0.94 $\pm$ 0.45
Creatinine after 12-18h from JC administration [mg/dL]	0.87 $\pm$ 0.45
RBC baseline [x 10 ⁶ /μL]	4.67 $\pm$ 0.46
RBC after 12-18h from JC administration [x 10 ⁶ /μL]	4.3 $\pm$ 0.5
HGB baseline [g/dL]	13.9 $\pm$ 1.4
HGB after 12-18h from IC administration [g/dL]	13.1 $\pm$ 1.54
HCT baseline %	40.6 $\pm$ 3.7
HCT after 12-18h from IC administration %	38.5 $\pm$ 4.3
BUN baseline [mg/dL]	37 $\pm$ 12.25
BUN after 12-18h from IC administration [mg/dL]	29.6 $\pm$ 10.5
Uric acid [mg/dL]	5.6 $\pm$ 1.6
TSH [μU/mL]	1.8 $\pm$ 1.43
TC [mg/dL]	174.6 $\pm$ 43.5
TG [mg/dL]	127.2 $\pm$ 69.4
Na ⁺ baseline [mmol/L]	139.4 $\pm$ 2.6
K ⁺ baseline [mmol/L]	4.04 $\pm$ 0.35
BUN- blood urea nitrogen, CABG- coronary artery by-pass graft, GFR glomerular filtration rate, HCT- hematocrit, HGB-hemoglobin, JC- iodine contrast, LDL- low density lipoprotein, PTCA- percutaneous transluminal coronary angioplasty, RBC-red blood cells, TC-total cholesterol, TSH-thyroid stimulating hormone.	

Table 2. Comparison of clinical characteristics between patients with or without reduction in serum creatinine concentration (only significant differences)

Parameter	Lack of SCR decrease (N=91)	Reduction of SCR (N=250)	p
Age [years]	59±8.5	61.5±8.5	p=0.015
RBC after 12-18 h from IC administration) [x 10 ⁶ /μl]	4.4±0.5	4.2±0.5	p=0.002
HGB at baseline) [g/dL]	14.2±1.3	13.8±1.4	p=0.006
HGB (after 12-18h from IC administration) [g/dL]	13.5±1.5	12.9±1.5	p<0.001
HCT (after 12-18h from IC administration) [g/dL]	39.7±4.2	38±4.2	p<0.001
Baseline BUN [mg/dL]	34.8±12.5	37.7±12.1	p=0.006
Baseline SCR [mg/dL]	0.88±0.6	0.95±0.7	p<0.001
Baseline CCR [ml/min]	115.8±37.8	94±33.1	p<0.001
GFR by CKD EPI baseline [mL/min/1.73 m ²]	91.3±18.3	79.3±19.1	p<0.001
GFR by MDRD baseline [mL/min/1.73 m ²]	92.3±24.3	76.8±20.9	p<0.001
Dysglycaemia %	53.5	41	p=0.04

Legend: BUN-urea, SCR-creatinine, CCR-creatinine clearance, GFR-glomerular filtration rate /Cockcroft Gault formula, CKD EPI (Chronic Kidney Disease Epidemiology Collaboration), MDRD formula (Modification of Diet in Renal Disease)/, HCT-hematocrit, HGB-hemoglobin, RBC red blood cells. P <0.05.

of myocardial infarction was present among 126 (35,3%) subjects. The characteristics of the study population with regard to laboratory parameters and medical records, classified by gender are shown in table 1. 55% of all subjects underwent coronary angioplasty. Patients were hydrated intravenously with 1637.4 ± 239.6 [mL] of saline. On average, patients received 110.2 ± 57.4 [mL] of contrast (during coronary angiography and/or percutaneous transluminal angioplasty). Intake of 1500 mL of liquids per person was recommended to all subjects; the actual intake was not measured.

Administered agents and medications

77.4% of the subjects were receiving angiotensin converting enzyme inhibitors (ACE - i), 11.7% angiotensin receptor blockers (ARBs), 86.2% beta- blockers (BB). Diuretics and statins were given to 43.5 and 94.4% of patients, respectively. Studied subgroups (i.e. groups in which contrast-induced deterioration in renal function was either present or absent), did not differ from each other in terms of pharmacotherapy.

Renal function vs. possible effect of dehydration

Control tests showed no reduction in serum creatinine in 26.7% of all respondents (91 patients). To exclude the improper hydration as a cause of no creatinine decrease the number of red blood cells as well as hemoglobin and hematocrit were analysed in blood donations collected during the study time points. No decrease in either red blood cells count (RBC), hemoglobin (HGB) or hematocrit (HCT) was observed in 46, 37 and 68 patients respectively (13.5% , 10.8%, and 20%). Among patients with no reduction in serum creatinine in control tests, concurrent reduction of RBC, HGB and HCT was observed respectively in 17 (18.6%) 13 (14.3%) and 24 (25.37%) patients. In the remaining 29 pts (46 pts-17pts) , 24 (37pts-13pts) and 44 (68pts-24pts) with no apparent reduction in RBC, HGB and HCT in control tests, serum creatinine level at the completion of the study was lower than at the baseline.

Comparison of patients with and without early post-procedural creatinine decrease

The comparison of clinical characteristics for continuous data, which differed between patients with and without early post-procedural creatinine decrease is shown in Table 2. There were 91 patients with no reduction and 250 individuals with a reduction in serum creatinine 12-18 hours after coronary angiography/angioplasty. No influence of media

Table 3. Summary of the parameters significantly associated with post-procedural lack of serum creatinine decrease

Parameter	Area under the curve	95% confidence interval	p
baseline serum creatinine	0.66	0.58-0.71	p<0.001
baseline creatinine clearance by C-G	0.68	0.62-0.75	p<0.001
GFR by CKD EPI	0.69	0.63-0.76	p<0.001
GFR by MDRD	0.70	0.64-0.77	p<0.001
GFR-glomerular filtration rate, C-G - Cocroft-Gault formula, CKD EPI (Chronic Kidney Disease Epidemiology Collaboration) formula, MDRD (Modification of Diet in Renal Disease) formula.			

Table 4. Summary of the results of univariate and multivariate logistic regression analysis

Variable	Univariate logistic regression			Multivariate logistic regression		
	Odds ratio	95% Confidence Interval	p value	Odds ratio	95% Confidence Interval	p value
Baseline serum creatinine <0.87 mg/dl	3.1	1.8-5.1	p<0.001	4.2	2.2-7.7	p<0.001
Baseline creatinine clearance by CG >86.77 [ml/min]	3.9	2.2-6.9	p<0.001	3.9	1.8-8.4	p=0.001
Baseline CKD EPI > 80.08 [ml/min/1.73m ²]	4.1	2-8.1	p<0.001	4.2	1.4-9	p=0.003
Baseline MDRD > 74.48 [ml/min/1.73m ²]	3.9	2.2-7	p<0.001	3.7	1.8-7.5	p<0.001
GFR-glomerular filtration rate, C-G (Cocroft-Gault) formula, CKD EPI (Chronic Kidney Disease Epidemiology Collaboration) formula, MDRD (Modification of Diet in Renal Disease) formula.						

contrast capacity and type of coronary procedure (simple angiography or angiography with angioplasty) on early creatinine shift was observed.

Factors related to the lack of creatinine decrease in control tests

Analysis of ROC curves showed that AUC for baseline creatinine, as well as creatinine clearance by C-G, GFR rate by CKD EPI and MDRD departed significantly from 0.5 with respect to association with the lack of creatinine decrease in control tests. AUC values were comparable for all analyzed parameters and remained within the range of 0.66 – 0.69. Cut-off points for parameters associated with the lack of creatinine decrease in control tests.

The following cut-offs were established based on the classification tree analysis:

- for baseline serum creatinine: value < 0.87 mg/dL;
- for creatinine clearance: value equal or higher than 86.77 mL/min;
- for GFR by CKD EPI: value equal or higher than 80.08 mL/min/1.73 m²;
- for GFR by MDRD: value equal or higher than 74.48 mL/min/1.73 m².

Assessment of risk for the lack of creatinine decrease in control tests in logistic regression

Results of univariate and multivariate logistic regression are shown in Table 3, 4 and 5. All pre-procedural parameters describing renal function identified patients at risk for post-procedural lack of creatinine decrease in control tests.

Discussion

Elective coronary angiography and percutaneous transluminal coronary angioplasty (PTCA) are commonly performed in clinical practice. The most common (observed among 3-5% of pts) non-cardiac side effect of these procedures is kidney damage [15-18]. The acute kidney injury (AKI) occurs when serum creatinine levels increases from the baseline by more

than 25% or 0.5 mg/dl during 24-48 h [3, 4]. The criteria of AKI are arbitral and the AKI thresholds are rather a consensus opinion of experts than reliable scientific method. This means that there is no established rule what level of serum creatinine increase is really significant for deterioration of renal function. Thus, the creatinine shift around 0.4 mg/dl will not be classified as an AKI, because the criteria is not met, while an even higher increase of SCR (more than 25% from baseline) may be clinically irrelevant. Additionally, the clinical criteria for CIN-AKI in the definition do not address the importance of prophylactic hydration.

Hydration and blood loss during angioplasty/coronary angiography should result in a decrease in hemoglobin level, RBC count (due to both dilution and loss) [1] and a decrease in the level of creatinine as a result of dilution. Thus, the increase in creatinine levels after administration of contrast medium in people who were preventively irrigated, should especially be worrying, for two reasons. Firstly, an increase

in creatinine after hydration often does not meet the criteria for CIN-AKI and therefore may mask the nephron damage. Secondly, failure to meet arbitrarily adopted thresholds for CIN-AKI results in the patient being considered healthy. Therefore, does not require any specific clinical investigation or practice. Fulfilling the temporary criteria (24-48 h) of contrast induced acute kidney injury by patients is even more difficult due to the fact, that people undergoing elective coronary intervention usually leave the hospital several hours after implementation of the procedure. Therefore, the essential SCR increase, which is necessary to recognize CIN-AKI, will occur in these individuals after discharge from the hospital, and thus it is often overloaded [19]. This is important issue from a clinical perspective, because undiagnosed CIN-AKI in 10% of the patients may lead to chronic renal failure, hemodialysis and premature death [15-18].

Table 5. Summary of multivariate analysis of lack of SCR decrease after angiography despite prophylactic peri-procedural hydration

Variable	Exp (B)	95% confidence interval	p
SCR <0.87 [mg/dl]	4.2	2.2-7.7	p<0.001
Male	2.2	1.2-4.2	p=0.015
Glucose disturbances	2.0	1.2-3.5	p=0.011
Chronic kidney disease	0.9	0.3-3	p=0.9
Heart failure	1.1	0.6-2.4	p=0.87
HGB	1	0.95-1.01	p=0.4
BUN	1	1-1.01	p=0.9
BMI	1	0.9-1	p=0.86
Age	0.9	0.9-1	p=0.34
CCR 86.77 [ml/min]	3.9	1.8-8.4	p=0.001
Glucose disturbances	1.7	1-3	p=0.051
Chronic kidney disease	0.7	0.3-3.1	p=0.9
Heart failure	1.3	0.6-2.6	p=0.5
HGB	1.11	0.9-1.3	p=0.3
BUN	1	0.9-1	p=0.6
BMI	0.96	0.9-1.02	p=0.2
Age	1	0.97-1.04	p=0.8
CKD EPI > 80.08 [ml/min/1.73m ²]	4.2	1.4-9	p=0.003
Glucose disturbances	1.7	1-2.9	p=0.045
Chronic kidney disease	1.6	0.4-6.3	p=0.5
Heart failure	1.2	0.62-2.5	p=0.6
HGB	1.1	0.94-1.4	p=0.2
BUN	1	0.96-1.02	p=0.6
BMI	1	0.94-1.06	p=0.99
Age	1	0.96-1.03	p=0.66
MDRD > 74.48 [ml/min/1.73m ²]	3.7	1.8-7.5	p<0.001
Glucose disturbances	1.7	1.1-2.9	p=0.05
Chronic kidney disease	1.2	0.4-4.1	p=0.74
Heart failure	1.25	0.6-2.5	p=0.53
HGB	1.17	0.95-1.4	p=0.13
BUN	1	1-1.01	p=0.76
BMI	1	0.94-1.05	p=0.89
Age	1	0.96-1.03	p=0.57

BMI-body mass index, SCR-creatinine, CCR-creatinine clearance, GFR-glomerular filtration rate /Cockcroft Gault formula, CKD EPI (Chronic Kidney Disease Epidemiology Collaboration), MDRD formula (Modification of Diet in Renal Disease)/, HGB-hemoglobin, BUN- blood urea nitrogen

The number of CIN-AKI will increase due to the fact that year by year the amount and types of percutaneous cardio-vascular procedures with the usage of CI increase (i.e. PTCA, transaortic valve replacement, thoracic endovascular aortic repair, endovascular aneurysm repair) [20, 21]. Thus, different tools are used for a better prediction of acute renal dysfunction including sophisticated clinical calculators or new biomarkers [20, 22, 23]. The fact that evaluation of serum creatinine or GFR can be a very effective detection method of early renal dysfunction was initially described by Ribichini et al [7]. In the current study we showed that the probability of serum creatinine increase in control tests was almost four times higher when baseline GFR exceeded 74.48 [mL/min/1.73 m²] for MDRD, 80.08 [mL/min/1.73 m²] for CKD EPI or when CCR was above 86.77 [mL/min]. The cut-off point for serum creatinine was less than 0.87 mg/dL (three fold increased risk for lack of creatinine decrease).

Surprisingly enough, in comparison to the patients with worse or already impaired renal function at the baseline, patients with initially better renal function are at higher risk of no creatinine decrease phenomenon. We are aware that the use of cut-off for SCR in the context of actual risk events assessment should not occur in such a small population like ours. Thus, the values in the group should be interpreted only as the results of the statistical model. However, the results have helped to make these clinical interpretations.

No published reports confirming association of initially better renal function with development of post procedural CI-AKI phenomenon were found in the available literature to our knowledge. However, we found literature showing the existence of the mechanism of direct and indirect (mediated by free oxygen radicals) toxicity of iodine contrast in kidneys [5]. High-osmolar iodine media increase oxygen requirements as well metabolic activity of kidneys and exacerbate imbalance between medulla and cortex, which induces hypoxia [5]. Ischemia is additionally promoted by vasoconstriction and blood flow reduction following administration of contrast, especially in the deeper parts of the outer medulla [5]. We may only speculate that the contrast administration is more dangerous for those with normal renal function, because previously non-toxic/ischemic tissue is more sensitive to any toxic/ischemic injury. It is probably due to the well-known but unaccounted for 'ischemic/toxic preconditioning', that the system is better prepared for ischemia/toxicities [24]. This may also explain why our population with the creatinine decrease during control tests, including older subjects with initially lower GFR and higher SCR, is probably better adapted to iodine contrast administration. However, further studies on extended population are required to confirm these findings.

It was also described that hydration among patients admitted to hospital may cause the phenomenon of no decrease in the serum creatinine next day, as shown in a control test, but the explanation of these is unknown. The possibility of this phenomenon playing a role in our study was also taken into account [3, 4].

Can the lack of early decline in SCR be considered as an important marker of renal function deterioration? The notion seems convincing, and it follows simple reasoning. Due to intravenous hydration, patient's intravascular volume increases, resulting in decrease in the creatinine concentration (subject to dilution). This effect should further reduce the negligible blood loss which occurs during angiography. Indeed, our analysis showed that in 89, 87% and 80% of patients final levels of red blood cells count (RBC), hemoglobin (HGB) and hematocrit (HCT) were lower than at the baseline. The creatinine levels were lower in 72% of patients. However, it should be mentioned that among all the 91 patients in whom no reduction of SCR was observed in the control tests, concurrent reduction of RBC, HGB and HCT was reported only in 16 (18%), 12 (13.4%) and 23 (25%) cases. In these patients absence of SCR decrease may be explained by an insufficient hydration before angiography. Lack of SCR reduction in remaining patients could be associated with other causes such as contrast-induced renal deterioration.

In addition, we noted a large disparity between the number of patients who displayed no change or even an increase in RBC, HGB compared to those with HCT increase. This is

probably due to the fact that HCT measurement depends not only on the RBC count but also on the size of red blood cells. It seems, therefore, that HCT is not an adequate parameter for such an analysis.

Limitations of the study.

The study is single center retrospective [25]. Another important limitation and disadvantage of our study is lack of the serum creatinine levels after 48-72 hours as well as after 30 days and 6 months after the procedure of contrast administration. There was no group to compare to, but this project was a pilot for a larger prospective study, which was conducted at that moment.

Formulas were used to estimate the creatinine clearance rate and GFR rather than direct measurements of these parameters. However, these formulas are recommended by contemporary guidelines and are currently used in clinical practice while direct measurement of creatinine clearance and GFR is not commonly performed [3, 4]. Analysed factors (multivariate regression) were the same for all models, although CCR, MDRD or CKD formulas use age, gender or weight in their patterns.

Conclusion

It was shown that the lack of creatinine decrease in control tests is more often observed among patients who have lower serum creatinine, better baseline creatinine clearance and GFR than subjects with creatinine decrease. These facts had not been reported previously in the literature. Precise GFR estimation should be carried out among all patients before and 12-18 h after all cardio-vascular procedures with IC use, because even in spite of prophylactic intravenous hydration, patients may develop contrast induced renal dysfunction.

Disclosure Statement

No conflict of interest is declared.

Acknowledgments

Special thanks to S. Nowak and K. Kempa for language and editing service.

References

- 1 Braunwald E, Gorlin R; Cooperative study on cardiac catheterization: Total population studied procedures employed and incidence of complications. *Circulation* 1968;37:III8-16.
- 2 Noto TJ Jr, Johnson LW, Krone R, Weaver WF, Clark DA, Kramer JR Jr, Vetrovec GW: Cardiac catheterization 1990: a report of the Registry of the Society for Cardiac Angiography and Interventions (SCA&I). *Cathet Cardiovasc Diagn* 1991;24:75-83.
- 3 http://www.kdigo.org/clinical_practice_guidelines/AKI.php
- 4 Lameire N, Kellum J, KDIGO AKI Guideline Work Group: Contrast-induced acute kidney injury and renal support for acute kidney injury: a KDIGO summary (Part 2). *Crit Care* 2013;17:205.
- 5 Bartorelli A, Marenzi G: Contrast-Induced Nephropathy. *J Interv Cardiol* 2008;21:74-85.
- 6 Erley CM: Does hydration prevent radiocontrast-induced acute renal failure? *Nephrol Dial Transplant* 1999;14:1064-1066.

- 7 Ribichini F, Graziani M, Gambaro G, Pasoli P, Pighi M, Pesarini G, Abaterusso C, Yabarek T, Brunelleschi S, Rizzotti P, Lupo A, Vassanelli C: Early creatinine shifts predict contrast-induced nephropathy and persistent renal damage after angiography. *Am J Med* 2010;123:755-763.
- 8 Burchardt P, Guzik P, Tabaczewski P, Synowicz T, Bogdan M, Faner P, Chmielarz-Sobocińska A, Palasz A: Early renal dysfunction after contrast media administration despite prophylactic hydration. *Int J Cardiovasc Imaging* 2013;29:959-966.
- 9 Task Force on Myocardial Revascularization of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS); European Association for Percutaneous Cardiovascular Interventions (EAPCI); Wijns W, Kolh P, Danchin N, Di Mario C, Falk V, Folliquet T, Garg S, Huber K, James S, Knuuti J, Lopez-Sendon J, Marco J, Menicanti L, Ostojic M, Piepoli MF, Pirllet C, Pomar JL, Reifart N, Ribichini FL, Schalij MJ, Sergeant P, Serruys PW, Silber S, Sousa Uva M, Taggart D: Guidelines on myocardial revascularization. *Eur Heart J* 2010;31:2501-2055.
- 10 Delanghe JR, Speeckaert MM: Creatinine determination according to Jaffe - what does it stand for? *NDT Plus* 2011;4:83-86.
- 11 Cockcroft DW, Gault MH: Prediction of creatinine clearance from serum creatinine. *Nephron* 1976;16:31-41.
- 12 Gault MH, Longerich LL, Harnett JD, Wesolowski C: Predicting glomerular function from adjusted serum creatinine. *Nephron* 1992;62:249-256.
- 13 Levey AS, Stevens LA, Schmid CH, Zhang YL, Castro AF 3rd, Feldman HI, Kusek JW, Eggers P, Van Lente F, Greene T, Coresh J; CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration): A new equation to estimate glomerular filtration rate. *Ann Intern Med* 2009;150:604-612.
- 14 Hunsicker LG, Adler S, Caggiula A, England BK, Greene T, Kusek JW, Rogers NL, Teschan PE: Predictors of the progression of renal disease in the Modification of Diet in Renal Disease Study. *Kidney Int* 1997;51:1908-1919.
- 15 Tavakol M, Ashraf S, Brene SJ: Risks and Complications of Coronary Angiography: A Comprehensive Review. *Glob J Health Sci* 2012;4:65-93.
- 16 Kennedy JW: Complications associated with cardiac catheterization and angiography. *Cathet Cardiovasc Diagn* 1982;8:5-11.
- 17 Kennedy JW, Baxley WA, Bunnell IL, Gensini GG, Messer JV, Mudd JG, Noto TJ, Paulin S, Pichard AD, Sheldon WC, Cohen M: Mortality related to cardiac catheterization and angiography. *Cathet Cardiovasc Diagn* 1982;8:323-340.
- 18 Johnson LW, Lozner EC, Johnson S, Krone R, Pichard AD, Vetrovec GW, Noto TJ: Coronary arteriography 1984-1987: a report of the Registry of the Society for Cardiac Angiography and Interventions. I. Results and complications. *Cathet Cardiovasc Diagn* 1989;17:5-10.
- 19 Burchardt P, Synowicz T, Jerzykowska S, Angerer D, Żurawski J: Early renal dysfunction after iodine contrast administration: new insight into the actual problem. *Kardiologia Polska* 2015;73:233-234.
- 20 Thongprayoon C, Cheungpasitporn W, Srivali N, Harrison AM, Gunderson TM, Kittanamongkolchai W, Greason KL, Kashani KB: AKI after Transcatheter or Surgical Aortic Valve Replacement. *J Am Soc Nephrol* 2015; pii: ASN.2015050577. [Epub ahead of print].
- 21 Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, Evangelista A, Falk V, Frank H, Gaemperli O, Grabenwöger M, Haverich A, Iung B, Manolis AJ, Meijboom F, Nienaber CA, Roffi M, Rousseau H, Sechtem U, Sirnes PA, Allmen RS, Vrints CJ; ESC Committee for Practice Guidelines: 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). *Eur Heart J* 2014;35:2873-2926.
- 22 Perrin T, Descombes E, Cook S: Contrast-induced nephropathy in invasive cardiology. *Swiss Med Wkly* 2012, DOI 10.4414/sm.w.2012.13608.
- 23 Altunoren O, Balli M, Eren N, Tasolar H, Arpacı A, Caglayan CE, Yavuz YC, Gungor O: Is Nebivolol really effective in preventing contrast induced nephropathy? *Kidney Blood Press Res* 2015;40:533-541.
- 24 Bonventre JV: Kidney ischemic preconditioning. *Curr Opin Nephrol Hypertens* 2002;11:43-48.
- 25 Hulley SB, Cummings SR, Browner WS, Grady DG, Newman TB (eds.): *Designing clinical research* (4th ED). Lippincott Williams & Wilkins, Philadelphia, USA 2013.