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Agreement between different eGFR equations in diagnosing contrast-induced acute kidney injury in patients undergoing elective coronary or peripheral angiography

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ABSTRACT

Background: In the present study, we aimed to compare CKD-EPI, MDRD, CKD-EPI creatinine-cystatin C equations and serum cystatin C and NGAL levels changes in assessing the occurrence of contrast-induced acute kidney injury (CI-AKI) in patients undergoing coronary and peripheral angiography and to evaluate the agreement between the CKD-EPI formula and the other parameters.

Methods: A cross-sectional study was performed in patients hospitalized with stable coronary artery disease and/or peripheral vascular disease, who underwent diagnostic and/or therapeutic invasive angiography using iodinated contrast agents. Standard laboratory parameters, NGAL, cystatin C levels, and eGFR were evaluated at admission and 48 hours after contrast substances exposure.

Results: Per different proposed definitions for CI-AKI, 7 patients (17.5 %) had a more than > 0.3 mg/dl increase in serum creatinine, 1 (2.5 %) had a > 25 % increase in serum cystatin C and 9 (22.5 %) had a > 25% increase in serum NGAL. The agreement between attributions based on CKD-EPI was excellent with MDRD (K coefficient 0.875), and modest with CKD-EPI creatinine-cystatin, which had also a modest agreement with MDRD (K coefficient 0.285). An increase in cystatinc C of more than 25% from baseline was not in concordance with a significant decrease in eGFR calculated with any equation, and the same was observed for NGAL.

Conclusions: In the present study, the performance of the CKD-EPI equation in diagnosing CI-AKI was not significantly better or worse than MDRD, CKD-EPI creatinine-cystatin, serum creatinine, NGAL or cystatin C increase.

Keywords: cystatin C-estimated glomerular filtration rate, neutrophil-associated lipocalin predictors, subclinical kidney injury

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INTRODUCTION

Post-contrast acute kidney injury is defined as a decrease in renal function after intravascular contrast administration, as estimated by an increase of 0.3 mg/dl or ≥1.5 x baseline serum creatinine levels measured 48-72 hours after the procedure [1] and is more frequently associated with intra-arterial contrast administration, like in coronary or peripheral angiography procedures, due to first-pass kidney exposure [2]. Even if serum creatinine is used to identify acute kidney injury, other important variables influence its concentration, among which the most important one is muscular mass, which is, in turn, dependent on the patient's age, weight, and gender. Also, creatinine increases relatively late after kidney injury, which may become apparent only when there is a more than 50% decrease in renal function [3]. Therefore, serum creatinine is not useful to identify early subclinical kidney dysfunction [4]. Even more, serum creatinine concentrations are not specific for different causes of kidney dysfunction such as contrast nephrotoxicity, dehydration or obstructive renal disease and are not a marker of tubular damage. This is why several equations have been proposed to include this variable to more precisely estimate the glomerular filtration rate (eGFR), such as CDK-EPI or MDRD.

Therefore, the estimated glomerular filtration rate is generally preferred for kidney function evaluation, and several formulas can be used. The European Society of Urogenital Radiology recommends using the CKD-EPI formula [1], but due to serum creatinine dynamics, it might not reflect the actual total glomerular filtration rate in

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patients with acute kidney injury. The same might also be true for other creatinine-based formulas, such as MDRD. There has been extensive interest in identifying more sensitive biomarkers for the prediction and diagnosis of subtle and early kidney lesions. Neutrophil gelatinase-associated lipocalin (NGAL) is a glycoprotein released from the distal nephron when there is acute kidney injury, and it is not significantly influenced by local hemodynamics. Its levels increase as early as 6 hours after percutaneous coronary interventions when there is contrast-induced nephropathy [5]. Similarly, another early biomarker for acute kidney impairment seems to be cystatin C, a low molecular-weight protein that is renally cleared and has a short half-time [6]. It is produced by all nucleated cells and undergoes free filtration in the glomeruli, being reabsorbed and metabolized in the proximal tubule. As NGAL and cystatin C are not affected significantly by age, gender or muscle mass, they might better reflect glomerular filtration rate than serum creatinine [7]. There is no accepted definition of subclinical contrast-induced nephropathy, but data from clinical studies showed that the substitution of serum creatinine with cystatin C or NGAL was associated with the diagnosis of twice as many cases of contrast-induced nephropathy as compared with the use of serum creatinine levels [8,9].

In the present study, we aimed to compare CKD-EPI, MDRD, CKD-EPI creatinine-cystatin C equations as well as serum cystatin C and NGAL level changes in assessing the occurrence of contrast-induced acute kidney injury in patients undergoing coronary and peripheral arteriography and to evaluate the agreement between the CKD-EPI formula and the other equations and serum parameters.

METHODS

Sample characteristics

This cross-sectional study included adult patients who underwent diagnostic and/or therapeutic invasive angiography using iodinated contrast agents for stable coronary artery disease and/or peripheral vascular disease, between January 2020 and November 2021. All patients provided written informed consent to participate in the present study. Patients with acute conditions such as infections were excluded from the study. The research protocol complied with the Declaration of Helsinki and it was approved by the Ethics Committee of the Emergency Institute for Cardiovascular Diseases and Transplantation Târgu Mureș and of George Emil Palade University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Romania.

Evaluated parameters

All patients considered eligible for the study were evaluated on admission. Data on gender, age, body height, and weight, as well as history of cardiovascular risk factors and comorbidities, such as previously diagnosed diabetes mellitus, arterial hypertension or chronic kidney disease, were recorded. Blood samples were collected from each patient on admission and serum creatinine was measured by Architect C4000 automated clinical chemistry analyser using the principle of Jaffe reaction. NGAL levels were measured using enzyme-linked immunosorbent assays on the ELISA Dynex DSX fully automated analyzer (DYNEX Technologies, Inc.; Chantilly, VA). Cystatin C values were assessed using latex-enhanced immunonephelometry. A second blood sample was collected for each patient 48 hours after contrast exposure evaluating serum creatinine, NGAL and cystatin C. Estimated glomerular filtration rates were calculated using CKD-EPI creatinine, CKD-EPI cratinine-cystatin and MDRD equations.

Contrast-induced acute kidney injury definition

Post-contrast acute kidney injury was defined as a 0.3 mg/dl increase in serum creatinine 48 hours after the procedure compared to the baseline measurement. A decrease of the estimated glomerular filtration rate below 60 ml/min/1.73 m^2 48 hours after contrast administration or a decrease of more than 25% in eGFR from baseline was considered acute kidney injury irrespective of the equation used. For serum NGAL and cystatin C, an increase of more than 25% from baseline at 48 hours post-contrast was considered acute kidney injury.

Statistical analysis

Continuous variables are presented as median and interquartile range and categorical data are expressed as numbers and percentages. All data were tested for normality using the Kolmogorov-Smirnov normality test. Based on the results of this test, parametric or non-parametric tests were used for between-group comparisons and correlation assessment. All tests were two-sided, and a p-value of less than 0.05 was considered statistically significant.

Weighted Cohen's kappa coefficient was used to assess the agreement in the classification of patients as having post-contrast acute kidney injury based on serum creatinine, eGFR with the three equations, NGAL and cystatin C. Concordance was defined as follows: K < 0.20 poor; 0.20–0.40 modest; 0.41–0.60 moderate; 0.61–0.80 good; >0.80 excellent.

Statistical analysis was performed using MedCalc for Windows, version 12.4.3.0 (MedCalc Software; Ostend, Belgium).

RESULTS

Study population characteristics

The study included 40 patients (mean age 64 ± 9 years; 32 % males). Thirty-eight patients (95.0 %) had a history of arterial hypertension, 18 patients (45 %) had a history of type 2 diabetes mellitus under treatment and 11 patients (27.5 %) had an eGFR (CKD-EPI equation) < 60 ml/min/1.73 m^2 on admission. Regarding the chronic treatment, 29 (72.5 %) were on angiotensin-converting enzyme inhibitors, 6 (15.0 %) on angiotensin receptor blockers, 21 (52.5 %) on loop-diuretics, and 11 (27.5 %) on metformin. Most patients (31, 77.5 %) received iomeprol solution, and the rest of them ioversol solution, with a median of 120 ml administered volume (100-160 ml).

Post-contrast acute kidney injury assessment with different measurements and equations

Serum creatinine, cystatin C, and NGAL measured before and 48 hours after contrast, were similar in the studied group (Table 1). Based on different proposed definitions for contrast-induced acute kidney injury, 7 patients (17.5 %) had a more than > 0.3 mg/dl increase in serum creatinine with a median increase of 0.53 mg/dl (0.34-1.04), 1 (2.5 %) had a > 25 % increase in serum cystatin C and 9 (22.5 %) had a > 25% increase in serum NGAL with a median increase of 41.9 % (34.7-66.9) for the latter.

Based on eGRF calculated with the CKD-EPI equation, 5 patients (12.5 %) had an eGFR decrease of > 25% from baseline and 2 patients (5%) with baseline normal eGFR had a clearance decrease below 60 ml/min/1.73 m^2. Using the CKD-EPI creatinine-cystatin C equation, none of the patients with baseline normal eGFR had an eGFR decrease below 60 ml/min/1.73 m^2 after contrast, but in 2 patients the eGFR decreased by > 25% from baseline. The MDRD equation identified 4 (10.0 %) patients with an eGFR decrease > 25 % from baseline, but only one patient with baseline normal MDRD eGFR had a post-contrast eGFR < 60 ml/min/1.73 m^2.

At baseline, there was excellent agreement between CKD-EPI and CKD-EPI creatinine-cystatin in identifying patients with eGFR below 60 ml/min/1.73 m^2 (K coefficient 0.817, 0.618-1.000) and good agreement between MDRD and both CDK-EPI and CKD-EPI creatinine-cystatin (K coefficient 0.661, 0.420-0.903 and 0.772, 0.498-0.946 respectively).

Agreement between different definitions for acute kidney injury after contrast

When performing the concordance analysis using serum creatinine increase as the reference, the highest, but modest agreement was found with MDRD (K coefficient 0.230). The agreement between attributions based on CKD-EPI was excellent with MDRD (K coefficient 0.875) and only modest with CKD-EPI creatinine-cystatin. The latter had also a modest agreement with MDRD (K coefficient 0.285) (Table 2). An increase in cystatin C of more than 25% from baseline was not in concordance with a significant decrease in eGFR calculated with any equation, and the same was observed for NGAL.

DISCUSSION

The main findings of the present study were that: (a) CKD-EPI and MDRD eGFR equations identified the highest number of patients with contrast-induced acute kidney injury after intra-arterial injection for coronary or peripheral angiography in stable patients, (b) there is an overall modest agreement between different eGFR equations in identifying patients with acute kidney injury after intra-arterial injection for coronary or peripheral angiography in stable patients, and (c) NGAL and cystatin C did not significantly increase in this cohort and they were not in agreement with eGFR.

Concordance between CKD-EPI and different eGFR equations in identifying patients with contrast-induced acute kidney injury

Serum creatinine and serum creatinine-derived eGFR, although far from ideal, remain the most widely used biomarkers of kidney function in clinical practice. Increase in serum creatinine and, consequently, decrease in eGFR have been identified as predictors of negative outcomes in patients receiving contrast agents for diagnostic or therapeutic purposes [10–12]. The definition of contrast-induced acute kidney injury relies on serum creatinine [1], but this has been repeatedly challenged

Table 1. Main characteristics of the study patients

	Before contrast	After contrast	*p* value
Serum creatinine (mg/dl)	1.01 (0.85-1.3)	1.06 (0.79-1.25)	0.92
NGAL (mg/l)	70.68 (53.41-93.58)	64.8 (47.17-83.24)	0.61
Cystatin C (mg/l)	1.06 (0.86-1.39)	1.01 (0.83-1.34)	0.32
CKD-EPI (ml/min/1.73 m^2)	77.0 (57.5-94.5)	85.5 (57.0-105.5)	0.13
CKD-EPI creatinine-cystatin C (ml/min/1.73 m^2)	85.0 (57.0-104.0)	85.0 (63.5-108.0)	0.04
MDRD (ml/min/1.73 m^2)	67.5 (53.0-84.2)	75.5 (51.5-93.5)	0.02

Quantitative data are expressed as median and interquartile range. *p* value was obtained using Wilcoxon test. NGAL – neutrophil gelatinase-associated lipocalin.

Table 2. Agreement in head-to-head comparison among eGFR equations and serum creatinine, NGAL, and cystatin C based definitions for contrast-induced acute kidney injury

	Before (n=40)	Mean eGFR before (ml/min/1.73 m²)	After (n=40)	Mean eGFR after (ml/min/1.73 m²)
CKD-EPI (< 60 ml/min/1.73 m²)	11 (27.5 %)	77.0 (57.5-94.5)	11 (27.5 %)	85.5 (57.0-105.5)
CKD-EPI CREA-Cystatin C (< 60 ml/min/1.73 m²)	12 (30.0 %)	85.0 (57.0-104.4)	9 (22.5 %)	85.0 (63.5-108.0)
MDRD (< 60 ml/min/1.73 m²)	15 (37.5 %)	67.5 (53.0-84.2)	13 (32.5 %)	75.5 (51.5-93.5)

	Weighted K coefficients (and 95% CI) after contrast				
	CKD-EPI	**CKD-EPI CREA-Cystatin C**	**MDRD**	**NGAL**	**Cystatin C**
Serum CREA increase (> 0.3 mg/dl)	0.181 (-0.176 to 0.540)	-0.086 (-0.188 to 0.014)	*0.230* (-0.134 to 0.595)	0.029 (-0.287 to 0.347)	-0.046 (-0.130 to 0.037)
CKD-EPI		*0.230* (-0.212 to 0.674)	***0.875*** (0.634-1.000)	-0.021 (-0.302 to 0.259)	-0.043 (-0.116 to 0.029)
CKD-EPI CREA-Cystatin C			*0.285* (-0.214 to 0.785)	0.108 (-0.180 to 0.398)	N/A
MDRD				0.017 (-0.268 to 0.304)	-0.041 (-0.108 to 0.025)
NGAL					-0.047 (-0.132 to 0.038)

Weighted Cohen's kappa coefficients [K (95% CI)]. Concordance was defined as follows: K < 0.20 poor; 0.20–0.40 modest; 0.41–0.60 moderate; 0.61–0.80 good; >0.80 excellent. Comparisons with moderate concordance are shown in italics and those with excellent concordance in bold italics. CREA- creatinine.

as it fails to assess the functional impact such as changes in urine output. There is a recommendation to use the CKD-EPI eGFR [1] in this setting, however, it is still unknown which equation would be better to use in different clinical scenarios to identify these patients optimally. Of all three tested equations, CDK-EPI, CKD-EPI creatinine-cystatin and MDRD identified the same patients with kidney dysfunction at baseline, only CKD-EPI and MDRD maintained a good agreement in finding patients with acute kidney injury after contrast. Our findings are in line with previously published data in an unselected cohort of patients admitted to a cardiology unit, with the highest agreement between CKD-EPI and MDRD, irrespective of age [13].

CKD-EPI creatinine-cystatin includes in its equation cystatin C, which is a marker of a more subtle nephron lesion not necessarily causing a decrease in urinary output, maybe explaining why there was no correlation with other equations. This hypothesis is further supported by the lack of agreement between any of the used eGFR equations and cystatin C or NGAL-based diagnosis of acute kidney injury.

Neutrophil gelatinase-associated lipocalin and cystatin C increase after contrast are not correlated with a significant decrease in eGFR

NGAL, a glycoprotein that is rapidly released into the bloodstream in response to renal tubular injury has been proposed as a biomarker for early and/or subtle kidney injury [9,14]. Unlike serum creatinine, NGAL increases even when the renal function is not significantly impaired, in the case of subclinical kidney injury [9]. A combination of markers for functional kidney damage, such as serum creatinine, and markers for structural kidney damage, such as NGAL, has been proposed to be used in order to stratify the risk of acute kidney injury [15]. Actually, when NGAL was used to evaluate the risk of developing contrast-induced acute kidney injury, the baseline measurement was inversely correlated with eGFR and was considered a predictor [16]. In our study, we evaluated the change in NGAL at 48h post-procedure and this variability did not prove to be useful in diagnosing contrast-induced acute kidney injury in our cohort.

Cystatin C is seen as a more accurate biomarker of kidney function than serum creatinine [17], as it is not influenced by muscle bulk, hemodynamics or anthropometric parameters and its value increases early after acute kidney injury. In our study, cystatin C alone or used in the CKD-EPI creatinine-cystatin equation did not show a diagnostic agreement with other measurements evaluated and this may be partly due to the 25% increase threshold that we used in our cohort, higher than previously published data [18]. Similar to data for NGAL, higher baseline cystatin C is predictive for post-contrast administration renal impairment, and not necessarily changes in cystatin C levels after contrast [19]. This makes cystatin C a promising predictive marker of contrast-induced kidney injury, and not necessarily a diagnostic marker.

Strengths and limitations

The present study investigated the agreement between several eGFR equations and changes in serum creatinine, NGAL and cystatin C in diagnosing contrast-induced

acute kidney injury. Although the present study did not identify the best method to diagnose it, it revealed two major findings: (a) that CKD-EPI and MDRD eGFR equations identified the highest number of patients with contrast-induced acute kidney injury after intra-arterial injection for coronary or peripheral angiography in stable patients and that (b) there is overall a modest agreement between different eGFR equations in identifying these patients. Finally, subclinical renal impairment was evaluated in the present study using NGAL and cystatin C, and their increase after contrast was not associated with significant decrease in eGFR. The major limitation of this study consists of the relatively small number of patients included, therefore the small number of patients with contrast-induced kidney injury.

CONCLUSIONS

In the present study, the performance of the CKD-EPI equation in diagnosing contrast-induced acute kidney injury in patients undergoing coronary or peripheral angiographies for stable conditions was not significantly better or worse than MDRD, CKD-EPI creatinin-cystatin, serum creatinine, NGAL or cystatin C increase. Our agreement analysis has important clinical implications since no actual data support the specific use of CKD-EPI formula for estimating GFR in this setting. Every formula is best used for the clinical scenario and the population it has been validated for, and no such equation was specifically designed to identify patients with contrast-induced acute kidney injury. Nevertheless, pending confirmation from future, larger scale studies, these results suggest that for this purpose CDK-EPI and MDRD eGFR can be used.

ABBREVIATIONS

CKD- chronic kidney disease

eGFR- estimated glomerular filtration rate

GFR- glomerular filtration rate

MDRD- Modification of Diet in Renal Disease

NGAL- neutrophil gelatinase-associated lipocalin

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AUTHORS' CONTRIBUTION

AS – Study design, statistical analysis, data management, data interpretation, writing draft

CS – Study design, data management, data interpretation, writing draft

TRN – data management, data interpretation

MO – Performed and interpreted the laboratory analyses

LD – Performed and interpreted the laboratory analyses

CONFLICT OF INTEREST

None to declare.

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