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Research Article

Feasibility of Using eGFR to Diagnose Contrast-Induced Nephropathy in Patients Undergoing Coronary Angiography

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ABSTRACT

Background: Contrast-induced nephropathy (CIN) is one of the common complications of coronary angiography (CAG). The changes of serum creatinine (Scr) before and after angiography were used to diagnose CIN in the world. But Scr is not a sensitive index to reflect early renal dysfunction. Estimated glomerular filtration rate (eGFR) is a comprehensive indicator to evaluate renal function but does not have accepted standard to diagnose CIN till now. This study aimed to investigate the feasibility of using eGFR to diagnose CIN in patients undergoing CAG.

Methods: We included 300 coronary heart disease (CHD) patients who underwent CAG. Their demographics and basal renal function were recorded. Changes of Scr and eGFR before and after CAG were compared at the same time. Logistic regression was used to find independent influence factors of CIN. Receptor operating characteristic (ROC) curve was used to find the optimum cut-off value of eGFR for diagnosing CIN.

Results: Among 300 patients with CHD, 64 (41 males and 23 females) of them were affected by CIN after CAG, with a total incidence of 21.3%. Among 271 patients whose Scr were normal (< 133 μmol/L) before CAG, 109 (40.2%) of them with impaired eGFR (< 90 ml/min/1.73m²). Patients had normal eGFR before CAG were less likely to develop CIN than those with normal Scr (15.4% vs 20.7%, $P < 0.05$). Logistic regression analysis showed that men, diabetes, multivessel lesion and eGFR were independent factors of CIN. ROC curve showed that the optimum cut-off value for diagnosing CIN was eGFR decrease by 22.5% after CAG (sensitivity = 98.4%, specificity = 98.3%, AUC = 0.973, 95%CI: 0.942-1.000, $P = 0.000$).

Conclusions: eGFR is an independent factor of CIN, which is more sensitive than Scr in reflecting early renal dysfunction. Using eGFR to diagnose CIN is feasible in the clinic, but the cut-off value still needs to be confirmed by large scale clinical trials.

Introduction

Coronary heart disease (CHD) has become the leading cause of death worldwide [1-2]. Coronary angiography (CAG) is the gold standard in the diagnosis of CHD [3]. Although the use of hypotonic non-ionic contrast agents can reduce renal damage, contrast-induced nephropathy (CIN) has significantly increased in recent years because of CAG being widely promoted. It is reported that about 4.4-11.3% of patients undergoing CAG will develop CIN, depending on populations, baseline risk factors and definitions [4-6].

The changes of serum creatinine (Scr) before and after CAG were used to diagnose CIN in the clinic [7]. However, Scr is not a perfect marker to reflect early renal dysfunction, because of its low sensitivity [8-10]. Before any Scr increase can be observed, more than 50% of glomerular

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filtration rate (eGFR) reduction occurr [11]. Thus, Scr to diagnose CIN may delay the optimal treatment time. Although eGFR is a sensitive index for early renal impairment, there is no specific standard to diagnose CIN by eGFR in the world till now. In this study, we aimed to investigate the feasibility of using eGFR to diagnose CIN and try to find out an optimum value.

Methods

I Study Design and Data Collection

This was a single-center observational, descriptive and analytical study that retrospectively analyzed the hospital record of a historical cohort of patients who underwent CAG between January 2015 and December 2016 in the People's Hospital of Xinyi. Patients with malignant tumors, obstructive nephropathy, renal artery stenosis, renal toxic medicine intake, hepatic diseases, and baseline renal function stage more than III, according to Scr, were excluded. Ethics Committees in Sun Yat-sen Memorial Hospital and People's Hospital of Xinyi approved this study. 300 patients undergoing CAG were included in our study. By checking the electronic database of patients' medical records, data of demographic, clinical, and laboratory test before and after CAG were collected. eGFR was calculated retrospectively using the simplified MDRD formula [$eGFR=186\times(Scr)^{-1.154}\times(Age)^{-0.203}\times(0.742$ if female)] [12, 13]. Changes of Scr and eGFR before and after CAG were compared at the same time.

II Definitions

CIN was diagnosed according to the K/DIGO guideline, which is the latest criteria. CIN was defined as an acute decrease in renal function after the exposure to contrast media, characterized by an absolute increase of ≥ 44.2 umol/L (≥ 0.5mg/dl) or ≥ 25% in Scr compared with baseline in the subsequent 48-72h, not explained by other causes [14]. Hypertension can be diagnosed by measuring SBP ≥ 140mmHg and/or DBP ≥ 90mmHg three times on different days without using antihypertensive drugs, or a history of hypertension diagnosis. Diabetes is defined as fasting blood glucose equal to or greater than 7.0 mmol/L, or decreased blood glucose with medication, or a history of diabetes diagnosis. Stroke is defined as cerebral infarction and/or cerebral hemorrhage with a history of more than 3 months. Heart failure included cardiac function NYHA or Killip class II ~ IV. Multivessel lesions indicated by CAG confirmed stenosis of at least two vessels in the left main coronary artery, left anterior descending branch, left circumflex branch and right coronary artery with stenosis of 70%.

III Cardiac Catheterization with CAG

Cardiac catheterizations with selective CAG were performed with standard techniques [15]. The results were recorded in both standard reports and digital imaging. Two interventional cardiologists who were unaware of the characteristics clinical, evaluated the CAG images independently. Both the anatomic feathers and the Gensini score were recorded.

IV Statistical Analysis

The normal-distributed quantitative data were expressed as mean value ± standard deviation and compared using the Students *t* test. Otherwise, median (25th～75th) was used and compared with Mann-Whitney U test. Categorical data were presented as absolute values and percentages and compared using chi square or Fishers exact test. Logistic regression model (enter), univariate and multivariate analyses were used to compare demographic, clinical and echocardiographic parameters to CIN. All variables in univariate analysis with P value < 0.20 entered multivariate analysis. The variables with P value < 0.05 were in the final model. Receptor operating characteristic (ROC) curve was used to find the optimum cut-off value of eGFR for diagnosing CIN. The SPSS software package for Windows 22 (IBM Corporation, New York, USA) was used for all statistical analysis. The significance was established at 2-tailed $P < 0.05$.

Results

I Baseline Characteristics of Study Population

As shown in (Table 1), a total of 300 patients were enrolled, including 223 (74.3%) males and 77 (25.7%) females. After a case-by-case analysis and a precise application of the CIN definition, 64 (41 males and 23 females) of them were affected by CIN after CAG, with a total incidence of 21.3%. In the CIN group, diabetes mellitus, emergency intervention, heart failure, multivessel lesion, baseline glucose level, and NT-proBNP were significantly higher than those in the non-CIN group, while the number of male patients, the levels of albumin, rehydration therapy rate and eGFR were lower than those in non-CIN group (P <0.05, Table 1). There were no significant differences in age, body mass index, hypertension, stroke, hematocrit, dosage of contrast agent, blood lipid and biochemical indexes between CIN group and non-CIN group.

II Scr and eGFR to Assess Early Renal Function

i Analysis of eGFR Levels in Patients with Normal Scr

There were 271 patients (90.3%) whose Scr were normal (< 133 μmol/L) before CAG. However, 109 (40.2%) of them had impaired eGFR (eGFR < 90 ml/min/1.73 m^2). It suggests that Scr is not a sensitive marker of early renal impairment compared with eGFR.

ii Comparison of Postoperative CIN in Patients with Normal Scr and Normal eGFR

Among 271 patients whose Scr were normal (< 133 μmol/L) before CAG, 56 (20.7%) of them develop CIN. Among 162 patients whose eGFR were normal (more than 90 ml/min/1.73 m^2) before CAG, there were only 25 (15.4%) of them develop CIN. Patients with normal eGFR before CAG were less likely to develop CIN than those with normal Scr ($P < 0.05$, Table 2).

III eGFR is an Independent Indicator of CIN

Logistics regression analysis was used to find the independent influencing factors of CIN. It showed that men and eGFR were protective factors for CIN, while diabetes and multivessel lesion were risk factors for CIN (Table 3).

IV The Optimum Cut-off Value of eGFR to Diagnose CIN

The ROC curve showed that the optimum cut-off value for diagnosing CIN was the decrease in eGFR by 22.5% after CAG (sensitivity = 98.4%, specificity = 98.3%, AUC = 0.973, 95%CI: 0.942-1.000, *P* = 0.000, (Figure 1A）. Also, we tried to find out if the absolute value of eGFR decrease will be more suitable than the relative value of decrease in diagnosing CIN. The ROC curve showed that the optimum cut-off value for diagnosing CIN was the absolute decrease in eGFR by 15.6 ml/min/1.73m² after CAG (sensitivity = 89.1%, specificity = 86.9%, AUC = 0.934, 95%CI: 0.898-0.971, *P* = 0.000, Figure 1B). Thus, using the relative changes of eGFR to diagnose CIN is better than using absolute changes.

Table 1: Demographic and clinical characteristics of the study population, according to development of CIN.

	non-CIN (n=236)	CIN (n=64)	*P*
Male，n(%)	182(77.1%)	41(64.1%)	0.010
Age，(years)	63.1±11.2	64.8±9.5	0.141
BMI，（Kg/m^2）	24.2±2.9	24.4±3.3	0.333
Hypertension，n(%)	122(51.7%)	33(51.6%)	0.110
Diabetes，n (%)	29(12.3%)	15(23.4%)	0.010
Stroke，n (%)	21(8.90%)	7(10.9%)	0.160
Emergent intervention，n (%)	52(22.0%)	22(34.4%)	0.020
Heart failure，n (%)	154(65.3%)	49(76.6%)	0.030
Multivessel lesion，n (%)	183(77.5%)	55(85.9%)	0.050
Hematocrit，n (%)	41.0±6.5	40.2±6.6	0.194
Contrast agent dose, (ml)	126.8±61.9	131.3±60.6	0.305
Rehydration therapy，n (%)	185(78.4%)	43(67.2%)	0.020
Albumin，(g/L)	40.3±3.8	38.9±4.9	0.009
BUN，(mmol/L)	5.98±2.69	6.04±2.90	0.441
Serum creatinine，(μmol/L)	87.5±49.4	92.4±28.3	0.154
Uric acid，(μmol/L)	362.2±99.83	382.3±137.4	0.137
Cystatin，(mg/L)	0.99±0.39	1.01±0.40	0.345
AST，(U/L)	36.6 (21.5-146.9)	78 (23.9-212.0)	0.324
TBIL，(μmol/L)	10.3±5.89	11.4±7.36	0.104
Potassium，(mmol/L)	3.88±0.53	3.95±0.62	0.186
Calcium；，(mmol/L)	2.21±0.20	2.18±0.18	0.241
Phosphorus，(mmol/L)	1.06±0.34	1.08±0.32	0.386
Glucose，(mmol/L)	7.82±4.80	9.29±6.76	0.025
CHOL，(mmol/L)	5.38±1.28	5.44±1.51	0.383
Triglyceride，(mmol/L)	1.95±1.48	1.73±1.04	0.140
LDL-C，(mmol/L)	3.26±1.02	3.20±1.07	0.331
NT-proBNP，(pg/ml)	176 (70.6-504)	275 (85.7-580)	0.017
eGFR(simplified MDRD formula)，(ml/min/1.73m²)	89.8±33.9	79.0±24.5	0.002

CIN：contrast-induced nephropathy; BMI：body mass index；BUN：Blood urea nitrogen; AST：aspartate aminotransferase；TBIL：total bilirubin；CHOL：total cholesterol; LDL-C：low density lipoprotein cholesterin; NT-proBNP：N-terminal pro brain natriuretic peptide; eGFR：glomerular filtration rate; simplified MDRD formula：$eGFR=186\times(Scr)^{-1.154}\times(Age)^{-0.203}\times(0.742\ If\ Female)$.

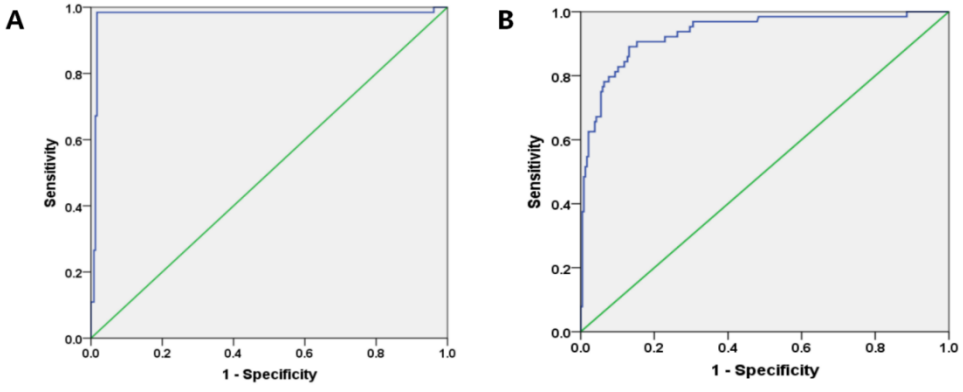

Figure 1: ROC curve of using eGFR to diagnose CIN.

A) The optimum cut-off value for diagnosing CIN was a decrease in eGFR by 22.5% after CAG (sensitivity = 98.4%, specificity = 98.3%, AUC = 0.973, 95%CI: 0.942-1.000, *P* = 0.000).

B) The optimum cut-off value for diagnosing CIN was a decrease in absolute value of eGFR by 15.6 ml/min/1.73m² after CAG (sensitivity = 89.1%, specificity = 86.9%, AUC = 0.934, 95%CI: 0.898-0.971, *P* = 0.000).

Table 2: The different prevalence of CIN between patients with Baseline normal Scr or Baseline normal eGFR after CAG.

	Baseline normal Scr（n=271）	**Baseline normal eGFR（n=162）**
CIN，n（%）	56（20.7%）	25（15.4%）
non-CIN，n（%）	215（79.3%）	137（84.6%）

CIN：contrast-induced nephropathy; eGFR：glomerular filtration rate

Table 3: Risk factors for CIN by logistics regression analysis.

	Univariate analysis			Multivariate analysis		
	OR	95% CI	*P*	OR	95% CI	*P*
Male	0.529	0.292-0.958	0.036	0.411	0.188-0.89*	0.026
Emergent intervention	1.853	1.017-3.380	0.044	1.458	0.416-5.116	0.556
Multivessel lesion	1.412	0.973-2.049	0.129	1.269	1.024-5.360	0.044
Rehydration therapy	0.547	0.295-1.015	0.050	1.104	0.322-3.781	0.875
Albumin	0.532	0.223-1.323	0.179	0.549	0.199-1.518	0.248
Scr	1.701	1.105-2.793	0.016	1.106	0.647-3.056	0.389
potassium	1.291	0.913-1.824	0.183	1.406	0.639-3.092	0.055
Glucose	1.367	1.026-1.822	0.033	1.876	0.777-4.530	0.162
NT-proBNP	1.223	1.004-1.497	0.046	1.159	0.911-1.475	0.230
eGFR(simplified MDRD formula)，(ml/min/1.73m²)	0.574	0.348-0.852	0.006	0.226	0.193-0.558	0.002
Hypertension	0.487	0.849-2.803	0.166	0.370	0.734-2.568	0.323
Diabetes	2.336	1.61-1.835	0.048	2.640	1.098-3.519	0.048
Heart failure	1.331	0.994-1.782	0.050	1.564	1.097-0.770	0.608
Hematocrit；	0.720	0.501-1.036	0.076	0.793	0.510-1.231	0.301
Age	1.179	0.874-1.546	0.299	--	--	--
Stroke	1.062	0.408-2.762	0.902			
BMI	1.186	0.682-2.060	0.547			
Contrast agent dose	1.204	0.682-2.126	0.521			
BUN	1.312	0.695-2.477	0.402			
Uric acid	0.823	0.455-1.487	0.518			
Cystatin；	1.116	0.882-1.342	0.297			
AST	1.353	0.776-2.359	0.287			
TBIL	1.030	0.715-4.184	0.724			
Calcium	0.913	0.703-1.186	0.494			
Phosphorus；	1.037	0.577-1.767	0.699			
CHOL	1.057	0.691-1.495	0.587			
Triglyceride；	1.153	0.955-1.244	0.682			
LDL-C	0.753	0.433-1.309	0.315			

CIN：contrast-induced nephropathy; BMI：body mass index；BUN：blood urea nitrogen; Scr：serum creatinine; AST：aspartate aminotransferase; TBIL：total bilirubin; CHOL：total cholesterol; LDL-C：low density lipoprotein cholesterol ; NT-proBNP：N-terminal pro brain natriuretic peptide; eGFR：estimated glomerular filtration rate; simplified MDRD formula：$eGFR=186\times(Scr)^{-1.154}\times(Age)^{-0.203}\times(0.742\ Female)$.

Discussion

I The Value of Scr and eGFR in Evaluating Early Renal Impairment

Early identification of CIN is of great significance to patients, but there are many difficulties. Diagnostic methods based on Scr have proved to be extremely limited. Scr and eGFR are both widely recognized indicators of renal function [16]. But Scr levels can be directly obtained by blood testing, while eGFR needs to be calculated by a complex formula; So eGFR is not as widely used as Scr. Most physicians are accustomed to using Scr to assess the level of renal function in the clinic. However, Scr is not sensitive enough to reflect the early renal dysfunction of the patient, due to the influence of age, sex, activity, weight, race and other factors [17]. Scr is not a perfect marker for reflecting renal function, because reductions of more than 50% in eGFR may occur before any increase in Scr is observed [11]. Therefore, the diagnosis and treatment of renal injury will be delayed. Although it's complicated to calculate eGFR, but because eGFR comprehensive considerate many factors that influence renal function, e.g. gender, age, Scr, race, BUN, ALB, etc., eGFR can much more fully to reflect the real level of renal function.

A study of 3,782 women with hypertension showed that about 50% of patients with a decrease in eGFR were within the normal range of Scr [18]. It is reported that Scr and BUN can still be in normal range in the early stages of renal impairment, when eGFR drops to about 80%. Our study also found that nearly 40% of CHD patients' Scr was within the normal range, with a decreased eGFR, which is consistent with the literature. Our study also found that patients with the normal eGFR had a lower proportion of CIN than those with normal Scr. If patients with normal Scr are considered with normal renal function before CAG and

didn't take any necessary preventive measures, the renal function will be further harm after CAG, thus easy to develop CIN. That why we believe eGFR is a better indicator of early renal impairment than Scr and suggested to use eGFR to evaluate the renal function of CHD patients before and after CAG in order to predict the risk of CIN and cardiovascular events.

II Analysis of the Renal Impairment Related Factors in CHD Patients

With the general development of coronary intervention techniques, the number of CIN is increasing. According to recent studies, the incidence of CIN caused by CAG was as high as 0.6% ~ 2.3% in patients with normal renal function, while the incidence of CIN caused by PCI was 3.3% ~ 14.5%. However, the incidence of CIN in patients with chronic kidney disease, chronic renal insufficiency, advanced age, diabetes, and heart failure is significantly increased, which can be as high as 25% - 50% [19-21]. It is reported that the related independent risk factors of CIN are advanced age, application of intra-aortic balloon pump (IABP), renal insufficiency, low hematocrit and diabetes [22-25]. Other scholars believe that the volume of contrast, congestive heart failure, hypotension or low blood volume, hypoalbuminemia and being female are also the risk factors for CIN [26, 27]. A multicenter prospective observational study of 906 patients with cardiac catheterization showed that reduced eGFR is an independent risk factor for CIN after cardiac catheterization [28]. In this study, the incidence of CIN were 21.3%, consistent with the previous report. Logistics regression analysis showed that men, diabetes, multi-vessel coronary artery disease and preoperative levels of eGFR are independent factors of CIN, diabetes and multi-vessel coronary artery disease are risk factors of CIN, while men and high eGFR are protective factors of CIN.

III The Feasibility Study on the Diagnosis of CIN by eGFR Determination and its Optimum Cut-off Value

A study of 860 patients with cardiac catheterization showed that change in eGFR ≤-1.1 mL/min/1.73 m^2 was a powerful independent predictor of CIN on the day following cardiac catheterization [16]. Our study found that eGFR was sensitive to early renal impairment in CHD patients. We used the current CIN diagnostic criteria as the gold standard and adopted the simplified MDRD formula to calculate eGFR. ROC curves found that when eGFR decreased 15.6 ml/min/1.73m^2, the sensitivity to diagnose CIN was 0.891, with the specificity of 0.869, and the AUC was 0.934. But if we use the relative changes of eGFR to diagnose CIN the optimum cut-off value was eGFR decrease 22.5% after CAG, and the sensitivity is 0.984, with the specificity of 0.983, AUC is 0.973. Thus, maybe using the relative changes of eGFR to diagnose CIN is better than using absolute changes. But there is no relevant criteria of eGFR to the diagnose CIN till now, and the effect of cut-off values on clinical promotion is uncertain. Due to the small number of cases in this study, the optimum cut-off value of eGFR in diagnosis of CIN still needs to be explored and confirmed by further large-scale prospective clinical studies.

Our study has some limitations. First, as a single-centre study with limited sample size, the evidence may not be as strong as that provided by a larger scale, multicenter trial study. The research conclusions only represented the actual situation of the samples in this study. Second, it is a retrospective observational study, and there will inevitably be some bias in the data.

Conclusion

This study demonstrated that eGFR is more sensitive than Scr in reflecting early renal dysfunction. It is an independent factor of CIN development after CAG. The optimum cut-off value of eGFR for diagnosing CIN was found to be a decrease of 22.5%. We should pay more attention to patients with impaired eGFR (< 90 ml/min/1.73m^2) before they use contrast media and take precautions to prevent the development of CIN in these patients. Using eGFR to diagnose CIN is feasible in the clinic, but the cut-off value still need to be confirmed by large scale clinical trials.

Conflicts of Interest

None.

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