



RESEARCH ARTICLE

Urinary neutrophil gelatinase-associated lipocalin predicted to contrast-associated acute kidney injury after planned percutaneous coronary intervention in elderly patients

Toan Nguyen Duy^{1,2} | Quyen Dao Bui Quy³ | Diem Ho Viet Le⁴ | Khoa Le Ha⁵ |
Dung Nguyen Huu⁶ | Kien Nguyen Trung^{1,2}  | Duy Tran Van^{1,2} |
Oanh Nguyen Oanh^{1,2} | Thuc Luong Cong^{1,2} | Hung Tran Duc^{1,2} | Thang Le Viet^{1,2} 

¹Military Hospital 103, Hanoi, Vietnam

²Vietnam Military Medical University, Hanoi, Vietnam

³Cho Ray Hospital, Ho Chi Minh, Vietnam

⁴Link-02 Clinic, Da Lat, Lam Dong, Vietnam

⁵Hanoi Medical University, Hanoi, Vietnam

⁶Bach Mai Hospital, Hanoi, Vietnam

Correspondence

Thang Le Viet, Military Hospital 103, Vietnam Military Medical University, 261 Phung Hung, Ha Dong, Hanoi, Vietnam.
Email: lethangviet@yahoo.co.uk

Abstract

Aim: To determine the proportion of contrast-associated acute kidney injury (CA-AKI) after percutaneous coronary intervention (PCI) and the predictive value of urine neutrophil gelatinase-associated lipocalin (uNGAL) for CA-AKI in elderly patients with chronic coronary artery disease.

Methods: A total of 509 patients who had planned percutaneous coronary intervention (mean age was 63.58 ± 11.63 years and 63.3% of males) were divided into two groups: group 1 ($n = 153$; elderly patients) with ≥ 70 years old and group 2 ($n = 356$) with < 70 years old. Urine NGAL was measured by the ELISA method. Clinical and laboratory data were collected on the day before intervention. CA-AKI was defined based on Kidney Disease: Improving Global Outcomes criteria.

Results: The ratio of CA-AKI in group 1 was 23.5% which was higher than that of group 2 (8.7%) with a p -value < 0.001 . Urine NGAL level in group 1 was significantly higher than that of group 2 [31.3 (19.16–55.13) ng/ml vs. 19.86 (13.21–29.04) ng/ml, $p < 0.001$]. At a cut-off value of 44.43 ng/ml, uNGAL had a predictive value for CA-AKI in all patients (AUC = 0.977, $p < 0.001$). Especially at a cut-off value of 44.14 ng/ml, uNGAL had a predictive value for CA-AKI in elderly patients (AUC = 0.979, $p < 0.001$).

Conclusions: The rate of CA-AKI after PCI in elderly patients was 23.5%. Urine NGAL before PCI had a good predictive value for CA-AKI in elderly patients with chronic coronary artery disease.

KEYWORDS

Chronic coronary artery disease, contrast-associated acute kidney injury, elderly patients, urine neutrophil gelatinase-associated lipocalin

Nguyen Duy Toan and Dao Bui Quy Quyen shared the first authorship.

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1 | INTRODUCTION

Coronary artery disease (CAD) is the most common cardiovascular disease caused by narrowing, constriction, or occlusion of the coronary arteries. To improve patient survival, percutaneous coronary intervention (PCI) is the most commonly applied reperfusion method in many countries, including Vietnam.¹ PCI relieves symptoms of myocardial ischemia and provides survival benefits in patients with coronary ischemic heart disease.² Contrast-associated acute kidney injury (CA-AKI) is an early complication after PCI. Depending on the study subjects, the rate of acute kidney injury can occur from 4.2% to 50%.³⁻⁵ CA-AKI after PCI is often associated with contrast drugs, hemodynamic instability, old age, pre-existing chronic kidney disease, or a combination of diabetes and hypertension.^{4,6} Old age is a risk factor for CA-AKI after PCI. Previous trials have shown that older age is associated with higher rates of PCI-related complications and worse short- and long-term prognosis in the presence of complications, including CA-AKI.^{7,8}

Despite the increasing heterogeneity of etiology, making a definitive and timely diagnosis of AKI remains challenging. Currently, the diagnosis of AKI is still based on elevated serum creatinine concentration or decreased urinary excretion. However, increased serum creatinine and decreased urine volume usually occur 48 hours after renal injury. Thus, it is necessary to use biomarkers that appear early from the time of renal damage in predicting AKI. Recently, urine Neutrophil Gelatinase-Associated Lipocalin (NGAL) has been evaluated as a valuable biomarker in predicting and diagnosing acute kidney injury in critical patients in intensive care units,⁹ patients with the first week after kidney transplantation¹⁰ as well as patients undergoing cardiovascular intervention.¹¹⁻¹³ NGAL, belonging to the lipocalin family, is a small protein with a molecular weight of 25kDa composed of 178 amino acids.¹⁴ Produced by neutrophils, plasma NGAL is freely filtered by the glomeruli and then completely reabsorbed by the proximal tubules.¹⁵

In this study, we wanted to investigate the frequency of CA-AKI occurrence in elderly patients after PCI and hypothesized that urine NGAL before intervention has a predictive value for CA-AKI in this subject.

2 | PATIENTS AND METHODS

2.1 | Patients

We included 674 patients with chronic coronary artery disease, indicated for planned percutaneous coronary intervention at two centers: Cardiovascular Center, Military Hospital 103, Hanoi, Vietnam, and Department of Cardiovascular Intervention, Tam Duc Hospital, Ho Chi Minh, Vietnam, from January 2016 to January 2017. We excluded patients <18 years of age or with previous percutaneous coronary intervention. The remaining 509 patients were provided written informed consent before participation in our study.

We collected all data on clinical characteristics and laboratory parameters at the baseline time of the study. The patient's 24-h-urine sample was collected on the day before the procedure. After 24 h, measure the urine volume, take 5 ml of urine to determine the NGAL level, then calculate the 24-h urine NGAL concentration. Urine NGAL was measured by the BioVendor Human Lipocalin-2/NGAL ELISA kit based on the sandwich enzyme immunoassay method. All patients were calculated glomerular filtration rate (eGFR) based on the MDRD formula.

We also collected fasting morning venous blood plasma to determine concentrations of CRP-hs, TnT-hs, ALT, AST, cholesterol, triglyceride, HDL-C, LDL-C, electrolyte, glucose, urea, and creatinine. EF% was calculated based on the patients' Doppler echocardiography results.

All patients were pre-treated with aspirin and oral clopidogrel 2-6 h before the procedure according to established protocol. The patient underwent percutaneous coronary angiography to assess coronary artery damage. Stent placement was performed when the degree of coronary stenosis was $\geq 70\%$. The operator decided on the type of coronary stent (bare-metal or drug-eluting stent). The duration, number, and type of stents were recorded for each patient. Contrast volume and type were also recorded, and the contrast drug volume/glomerular filtration rate ratio was calculated for each patient. Any complications occurring during or within the first 24 h of PCI, as well as the following days, were also recorded. All patients had their urine monitored, and serum creatinine (sCr) was measured 48 h after the procedure to detect AKI.

Acute kidney injury was defined based on Kidney Disease: Improving Global Outcomes (KDIGO) criteria.¹⁶ Patients were classified as the severity of AKI was graded as (1) Stage 1 in proportion to a 1.5-1.9-fold increase of sCr level or an acute rise in sCr of more than $26.5 \mu\text{mol/L}$ (0.3 mg/dl) within 48 h (2); Stage 2 in proportion to a 2.0-2.9 fold increasing of sCr level and (3) Stage 3 in proportion to a 3.0-fold increasing of sCr or $\text{sCr} \geq 353.6 \mu\text{mol/L}$ (4.0 mg/dl) or need for dialysis.

All 509 patients were divided into two groups: Group 1 ($n = 153$): Patients ≥ 70 years old, and Group 2 ($n = 356$): Patients < 70 years old, to find the role of uNGAL in predicting contrast-associated acute kidney injury after planned percutaneous coronary intervention in elderly patients.

2.2 | Statistical analyses

All the normal distribution continuous data were represented by mean and standard deviation and were analyzed by Student *t*-test. All the skewed distributions were described by median (25 percentile - 75 percentile), analyzed by Mann-Whitney *U* test and Kruskal-Wallis test. Categorical data were presented by the frequency with percentage and were analyzed using the chi-square test. Multivariable adjusted regression analysis was performed to identify the predictors of AKI. Receiver operating characteristic (ROC) curves with the area under the curve (AUC) was calculated to predict AKI from all patients

TABLE 1 Comparison of demographic and laboratory characteristics in group 1 and group 2

Clinical characteristics and laboratory parameters	Total (n = 509)	Group 1 (n = 153)	Group 2 (n = 356)	p
Ages (Years)	63.58 ± 11.63	77.22 ± 5.82	57.72 ± 8.04	<0.001
Number of males (n, %)	322 (63.3)	88 (57.5)	234 (65.7)	0.078
Hypertension				
• Yes (n, %)	145 (28.5)	87 (56.9)	58 (16.3)	<0.001
• No (n, %)	364 (71.5)	66 (43.1)	298 (83.7)	
Diabetic mellitus				
• Yes (n, %)	120 (23.6)	71 (46.4)	49 (13.8)	<0.001
• No (n, %)	389 (76.4)	82 (53.6)	307 (86.2)	
BMI (kg/m ²)				
• <18.5 (n, %)	19 (3.7)	12 (7.8)	7 (2.0)	0.001
• 18.5–22.9 (n, %)	149 (29.3)	52 (34)	97 (27.2)	
• ≥23.0 (n, %)	341 (67)	89 (58.2)	252 (70.8)	
• Mean	24.23 ± 3.34	23.36 ± 3.18	24.61 ± 3.34	<0.001
Anemia (n, %)	138 (27.1)	61 (39.9)	77 (21.6)	<0.001
Hemoglobin (g/L)	134.5 ± 18.95	126.91 ± 20.21	137.76 ± 17.42	<0.001
Creatinine (μmol/L)	89 (79–103)	89 (79–110)	89 (78.25–101)	0.254
CRP-hs (mg/L)				
• >5.0 (n, %)	112 (22)	40 (26.1)	72 (20.2)	0.139
• Median	2.8 (1.9–4.5)	2.9 (2.0–5.3)	2.8 (1.8–4.3)	0.117
TnT-hs (ng/L)				
• >14.0 (n, %)	356 (69.9)	114 (74.5)	242 (68)	0.141
• Median	41 (11.53–234.65)	49 (13.92–209.95)	38.3 (10.7–277.52)	0.458
ALT (UI/L)				
• >40.0	139 (27.3)	34 (22.2)	105 (29.5)	0.091
• Median	29 (21–43)	28 (19–39.5)	31 (22–45)	0.016
AST (UI/L)				
• >40.0	172 (33.8)	51 (33.3)	121 (34)	0.886
• Median	33 (25–47)	33 (25–45)	33 (25–48)	0.693
Cholesterol (mmol/L)				
• ≥5.2	156 (30.6)	44 (28.8)	112 (31.5)	0.544
• Median	4.56 (3.65–5.45)	4.38 (3.58–5.46)	4.76 (3.67–5.45)	0.107
Triglyceride (mmol/L)				
• ≥2.3	180 (35.4)	40 (26.1)	140 (39.3)	0.004
• Median	1.89 (1.28–2.74)	1.71 (1.14–2.30)	2.02 (1.32–3.1)	0.001
LDL-C (mmol/L)				
• ≥3.2	194 (38.1)	51 (33.3)	143 (40.2)	0.145
• Median	2.8 (2.1–3.5)	2.7 (2.1–3.4)	2.89 (2.1–3.54)	0.166
HDL-C (mmol/L)				
• ≤0.9	98 (19.3)	26 (17)	72 (20.2)	0.397
• Median	1.16 (0.98–1.36)	1.17 (0.99–1.36)	1.15 (0.96–1.36)	0.640
Lipid disorder (n, %)	366 (71.9)	92 (60.1)	274 (77)	<0.001
Na ⁺ (mmol/L)	137.54 ± 3.83	137.07 ± 4.6	137.74 ± 3.44	0.111
K ⁺ (mmol/L)	3.8 (3.55–4.04)	3.76 (3.56–3.98)	3.82 (3.54–4.05)	0.167
EF%				
• <50.0%	141 (27.7)	54 (35.3)	87 (24.4)	0.012
• Median	61 (45–70)	61 (40–69)	61 (50–71)	0.042

(Continues)

TABLE 1 (Continued)

Clinical characteristics and laboratory parameters	Total (n = 509)	Group 1 (n = 153)	Group 2 (n = 356)	p
Urine NGAL (ng/ml)	21.3 (14.31–36.07)	31.3 (19.16–55.13)	19.86 (13.21–29.04)	<0.001
eGFR (ml/min/1.73 m ²)				
• <60	218 (42.8)	125 (81.7)	93 (26.1)	<0.001
• Median	64.09 (47.87–78.87)	45.19 (36.64–56.84)	71.28 (59.55–85.68)	<0.001
Vc/eGFR ratio	1.83 (1.4–2.6)	2.53 (2.04–3.38)	1.59 (1.28–2.12)	<0.001
Number of stents				
• 1 (n, %)	317 (62.3)	102 (66.7)	215 (60.4)	0.004
• 2 (n, %)	111 (21.8)	39 (25.5)	72 (20.2)	
• 3 (n, %)	81 (15.9)	12 (7.8)	69 (19.4)	
• Mean	1.54 ± 0.75	1.41 ± 0.63	1.59 ± 0.79	0.008
AKI (n, %)	67 (13.2)	36 (23.5)	31 (8.7)	<0.001

Abbreviations: AKI, Acute Kidney Injury; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; BMI, Body Mass Index; CRP-hs, C-Reactive Protein-high sensitive; EF, Ejection Fraction; eGFR, estimated Glomerular Filtration Rate; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; NGAL, Neutrophil Gelatinase-associated Lipocalin; TnT-hs, Troponin T-high sensitive; Vc, Contrast Drug Volume.

p-value < 0.05 are indicated in italics.

and patients ≥70 years old. Statistical analysis was performed using Statistical Package for Social Science (SPSS) version 20.0 (Chicago, IL, USA). A *p*-value < 0.05 was considered significant.

3 | RESULTS

The results in Table 1 show that the ratio of hypertension, diabetes, and anemia in group 1 was significantly higher than in group 2, *p* < 0.001. Mean BMI, hemoglobin, and eGFR in group 1 were significantly lower than in group 2, *p* < 0.001. Opposite, uNGAL concentration, and Vc/eGFR in group 1 were significantly higher than in group 2, *p* < 0.001. Especially ratio of AKI in group 1 was higher than that of group 2, *p* < 0.001.

Multivariate logistic regression analysis in Table 2 shows that only a high Vc/eGFR ratio was an independent factor related to the occurrence of AKI in elderly patients.

Based on ROC curve model analysis, urine NGAL, Vc/eGFR ratio, eGFR, and age were good factors in predicting AKI in all patients after PCI, *p* < 0.001 (Figure 1), while urine NGAL, eGFR, and Vc/eGFR ratio were good factors in predicting AKI in elderly patients after PCI, *p* < 0.001 (Figure 2).

4 | DISCUSSION

4.1 | Ratio of contrast-associated acute kidney injury after planned percutaneous coronary intervention in elderly patients

For patients with chronic coronary artery disease, percutaneous coronary angiography is indicated for patients with stenosis ≥70% of coronary artery diameter. Iodinated contrast agents will be used

for percutaneous coronary angiography procedures to determine the extent of coronary artery damage and intervene when indicated. AKI in this group of patients may be related only to the contrast agent. However, some patients may also be associated with other features such as hemodynamic changes before and after intervention, chronic kidney disease status before the intervention, or heart failure degree. In this study, like some other authors, we use the term CA-AKI to show the heterogeneity in the etiology and multifactorial influence of AKI.^{17,18} The rate of AKI in our study was 13.2%, and the rate of AKI in ≥70 years old patients was higher than in the group of <70 years old ones (23.5% vs. 8.7%, *p* < 0.001, Table 1). Experimental evidence has shown that contrast agents reduce renal blood flow in the medulla, generate free oxygen radicals, and induce apoptosis of renal tubular cells, thereby causing kidney damage.^{19,20} Clinically, the elevation of serum creatinine has occurred in patients receiving contrast media.²¹ Elderly patients have a higher incidence of CA-AKI than younger adults, and older age is a risk factor for CA-AKI in patients after PCI.^{22–24} There are several reasons for the high rate of AKI in the elderly after PCI compared to that in young people: the elderly have more complex coronary artery lesions than the younger population, and older people will undergo more complicated procedures than was the case in the past.²⁵ In addition, aging causes changes in renal function (increased renal excretion of sodium and water).²⁶ Older people often decrease thirst, dehydration, or comorbidities with chronic diseases and more advanced vascular diseases such as long-term hypertension and diabetes (Table 1).

The results of our study show that the ratio of contrast volume/glomerular filtration rate is an independent factor related to AKI in older people undergoing PCI (OR: 7.599; 95% CI: 2.834–20.377; *p* < 0.001) (Table 2). The volume of contrast medium used in PCI has been implicated as the most critical factor in the induction of AKI.²⁷ The Vc/GFR ratio, a valuable indicator for developing

kidney disease, was also found in this patient population.²⁸ The mechanisms underlying AKI by contrast agents have not been fully elucidated. However, physiologically when contrast agents are injected intravenously or intra-arterial, they pass from the vascular compartment through the capillaries into the extracellular space. They are eliminated almost entirely through glomerular filtration, concentrated in the tubular lumen by water reabsorption, and then excreted in the urinary tract.²⁹ In the case of patients with a high Vc/eGFR ratio, the kidneys will not be able to eradicate the contrast. The contrast will stagnate in the renal tubules, causing damage to the renal tubular epithelial cells and/or stagnation in the kidney interstitium, causing acute interstitial nephritis and two AKI lesions.

TABLE 2 Multivariate logistic regression analysis of some clinical variables related to AKI in ≥70 years old patients

Variable	OR	95% CI	p
Female	0.68	0.274–1.69	0.407
Anemia	1.554	0.618–3.906	0.349
Hypertension	3.225	0.779–13.356	0.106
Diabetes	1.845	0.533–6.38	0.334
CRP-hs > 5.0 mg/L	0.441	0.14–1.386	0.161
EF < 50.0%	0.865	0.339–2.205	0.761
Vc/eGFR ratio > 3.7	7.599	2.834–20.377	<0.001

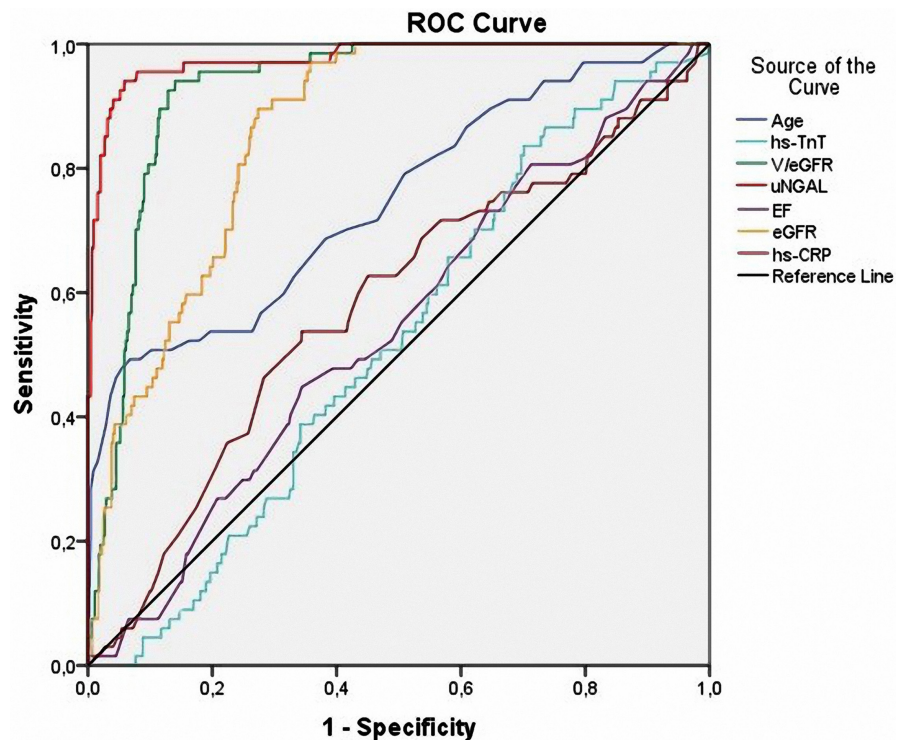
Abbreviations: CRP-hs: C Reactive Protein-high sensitive; EF: Ejection Fraction; eGFR: estimated Glomerular Filtration Rate; Vc: Contrast Drug Volume.
p-value < 0.05 are indicated in italics.

4.2 | Role of urine NGAL in predicting contrast-associated acute kidney injury after planned percutaneous coronary intervention in elderly patients

According to the results of our study, the Vc/eGFR ratio can be used to predict the development of CA-AKI in patients after PCI. In the total of 509 studied patients (Figure 1), a Vc/eGFR ratio > 2.61 was an essential limit in predicting the development of CA-AKI (AUC: 0.926, *p* < 0.001). Interestingly, in the elderly group, the Vc/eGFR ratio > 2.61 is also the limit to predict the development of CA-AKI, AUC = 0.832, *p* < 0.001 (Figure 2). Worasuwanarak S et al.³⁰ also reported the same study results as ours, with a Vc/eGFR ratio > 2.6 predicting CA-AKI in patients after PCI. After PCI, the mechanism of CA-AKI has not been precisely elucidated. Many factors are involved in the occurrence of CA-AKI in elderly patients. They can be divided into two groups: favorable patient characteristics including diabetes mellitus, hypertension, anemia, heart failure, and impaired renal function (these variables are more likely to occur in the elderly group, Table 1). Regarding PCI, the factors that facilitate CA-AKI development are an increased amount of contrast agent and a high Vc/eGFR ratio (these variables in the elderly group in our study also accounted for a higher proportion, Table 1).

Some authors have also used uNGAL to predict AKI in patients undergoing PCI or prior cardiovascular surgery.^{11–13} In this study, we also quantified the concentration of uNGAL in patients before the PCI. Analyzing the results showed that the concentration of uNGAL in the elderly group was higher than in the group of patients <70 years old, [31.3 (19.16–55.13) ng/ml vs. 19.86 (13.21–29.04), *p* < 0.001, (Table 1)].

FIGURE 1 Receiver operating characteristic (ROC) curves of Age, CRP-hs, TnT, EF%, eGFR, Vc/eGFR, and uNGAL predict AKI in all studied patients. (uNGAL: AUC = 0.977; *p* < 0.001; cut-off value = 44.43 ng/ml; Sensitivity = 94%; Specificity = 94.1%; Vc/eGRF ratio: AUC = 0.926; *p* < 0.001; cut-off value = 2.61; Sensitivity = 94%; Specificity = 86%; eGFR: AUC = 0.859; *p* < 0.001; cut-off value = 55.74 ml/min/1.73 m²; Sensitivity = 89.6%; Specificity = 72.6%; Age: AUC = 0.746; *p* < 0.001; cut-off value = 77.5 years old; Sensitivity = 49.3%; Specificity = 93.2%; hs-CRP: AUC = 0.58; *p* = 0.035; cut-off value = 2.15 mg/L; Sensitivity = 53.7%; Specificity = 65.6%)



Diagonal segments are produced by ties.

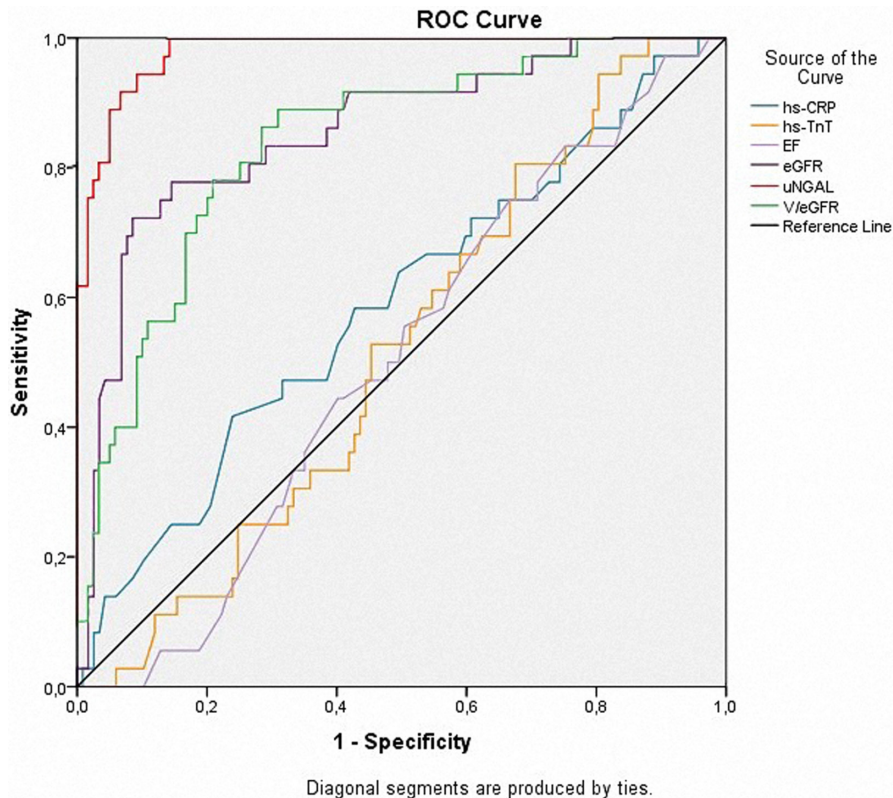


FIGURE 2 Receiver-operating characteristic (ROC) curves of CRP-hs, TnT, EF%, eGFR, Vc/eGFR, and uNGAL for predicting AKI in ≥ 70 years old patients). (Urine NGAL: AUC = 0.979; $p < 0.001$; cut-off value = 44.14 ng/ml; Sensitivity = 100%; Specificity = 85.5%; eGFR: AUC = 0.856; $p < 0.001$; cut-off value = 35.53 ml/min/1.73 m²; Sensitivity = 72.2%; Specificity = 91.5%; Vc/eGRF ratio: AUC = 0.832; $p < 0.001$; cut-off value = 2.61; Sensitivity = 88.9%; Specificity = 68.4%)

And especially, with uNGAL concentration > 44.43 ng/ml, there was a predictive value of CA-AKI in all patients in this study (AUC: 0.977, $p < 0.001$; Figure 1) and with concentrations uNGAL > 44.14 ng/ml had a predictive value of CA-AKI in elderly patients (AUC: 0.979, $p < 0.001$; Figure 2). Thus, uNGAL is a biomarker with a good predictive value of CA-AKI in all patients and elderly patients after PCI.

Although our research results have answered the research objectives, our study also had limitations: it had not clarified the factors of the PCI process to the occurrence of CA-AKI, and the role of uNGAL had not been clarified in predicting long-term outcomes of PCI in the elderly patients. In addition, the study did not have a healthy control group to describe the rate and extent of urinary NGAL elevation in the patient group.

5 | CONCLUSION

The rate of CA-AKI after PCI in elderly patients was 23.5%. Urine NGAL before PCI has a good predictive value for CA-AKI in elderly patients who received planned percutaneous coronary intervention.

AUTHOR CONTRIBUTIONS

Research idea and study design: Nguyen Duy Toan, Dao Bui Quy Quyen, Le Viet Thang. Data acquisition: Nguyen Duy Toan, Dao Bui Quy Quyen, Ho Viet Le Diem, Le Ha Khoa. Data analysis/interpretation: Nguyen Trung Kien, Tran Van Duy, Le Ha Khoa. Statistical analysis: Nguyen Trung Kien, Tran Van Duy. Supervision or mentorship:

Nguyen Huu Dung, Nguyen Oanh Oanh, Luong Cong Thuc, Tran Duc Hung, Le Viet Thang.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial, or otherwise.

DATA AVAILABILITY STATEMENT

Authors are able to provide additional relevant original data underpinning their research if requested by the Editor or reviewers.

INFORMATION PERTAINING TO WRITING ASSISTANCE

We do not receive any funded writing assistance to create this manuscript.

ORCID

Kien Nguyen Trung  <https://orcid.org/0000-0002-0333-2612>

Thang Le Viet  <https://orcid.org/0000-0002-2283-9988>

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How to cite this article: Nguyen Duy T, Dao Bui Quy Q, Ho Viet Le D, et al. Urinary neutrophil gelatinase-associated lipocalin predicted to contrast-associated acute kidney injury after planned percutaneous coronary intervention in elderly patients. *J Clin Lab Anal*. 2022;36:e24757. doi: [10.1002/jcla.24757](https://doi.org/10.1002/jcla.24757)