

Original Research

INCIDENCE OF CONTRAST-INDUCED ACUTE KIDNEY INJURY AND RELATED FACTORS IN PATIENTS ≥ 65 YEARS OLD WITH CHRONIC KIDNEY DISEASE AND ACUTE CORONARY SYNDROME AFTER PERCUTANEOUS CORONARY INTERVENTION

Chau Van Vinh¹, Pham Dinh Giang¹, Dinh Van Tin¹, Nguyen Van Tan¹²,*

1. Thong Nhat Hospital, Vietnam

2. Department of Geriatrics, University of Medicine and Pharmacy, Ho Chi Minh City Corresponding Author: Nguyen Van Tan, ☑ nguyenvtan10@ump.edu.vn

ABSTRACT: Contrast-induced nephropathy (CIN) is a common complication following percutaneous coronary intervention (PCI), particularly in older patients with chronic kidney disease (CKD) and acute coronary syndrome (ACS). CIN can lead to worsening renal function, increased mortality, and prolonged hospitalization. However, data on the incidence and associated factors of CIN in this high-risk population remain limited. To determine the incidence and associated factors of CIN in patients aged ≥65 years with CKD and ACS undergoing percutaneous coronary intervention (PCI). A prospective descriptive study enrolled 225 patients aged ≥65 years with CKD and ACS admitted to Thong Nhat Hospital from May 2024 to May 2025. Serum creatinine and estimated glomerular filtration rate (eGFR) were monitored before and up to five days post-PCI. CIN is defined as an increase in creatinine ≥25% or ≥0.5 mg/dL from baseline. Risk factors analyzed include the Mehran risk score, cardiovascular history, hemodynamic status, anemia, and contrast volume. CIN occurs in 13.8% of patients, with significantly higher incidence in CKD stage 4–5 compared to stage 3 (23.3% vs. 9.2%; p = 0.004). The CKD 4–5 group has a higher mean Mehran score (15.4 vs. 7.31; p = 0.001) and lower post-PCI eGFR (16.8 vs. 46.6 mL/min/1.73 m²; p < 0.001). Two patients (0.9%) require emergency dialysis, and in-hospital mortality is 1.8%, mostly among those with advanced CKD. Significant predictors of CIN include Mehran score ≥10, heart failure, anemia, hypotension, and higher contrast volume. CIN remains a relevant complication in elderly patients with CKD and ACS undergoing PCI, especially in those with advanced CKD. Early risk stratification, hemodynamic optimization, and careful management of contrast use are essential to reducing CIN in this high-risk group.

Keywords: contrast-induced nephropathy; acute kidney injury; chronic kidney disease; acute coronary syndrome; percutaneous coronary intervention; elderly patients

1. INTRODUCTION

Iodinated contrast agents are widely used in diagnostic and interventional coronary procedures, significantly improving treatment outcomes prognosis for cardiovascular patients. However, the use of contrast agents carries the risk of acute kidney injury (AKI), referred to as contrast-induced nephropathy (CIN). CIN is currently the third most common cause of hospitalacquired AKI and is associated with increased mortality, prolonged hospital stays, higher treatment costs, and an elevated risk of progression to end-stage renal disease (ESRD) [1].

The risk of CIN is heightened in individuals with underlying risk factors such as age ≥65 years, chronic kidney disease (CKD), diabetes mellitus, heart failure, hypovolemia, or exposure to high doses of contrast agents [2]. Among these, elderly patients with CKD are particularly vulnerable due to age-related decline in renal function combined with comorbidities such as atherosclerosis, failure, anemia, and complex coronary artery disease. Mehran et al. developed a risk prediction score for CIN, highlighting factors such as advanced age, low eGFR, diabetes, anemia, and contrast agent dose as significant contributors to CIN risk following percutaneous coronary intervention (PCI) [3].

Despite advancements in the prevention and management of CIN, the incidence of AKI after PCI remains high in elderly patients with CKD. In Vietnam, data on the incidence and risk factors of CIN in this patient group are limited. Therefore, this study aims to determine the incidence of CIN and its associated factors in patients aged ≥65 years with CKD and acute coronary syndrome (ACS) after PCI, thereby providing guidance for effective interventions and prevention of this complication.

2. SUBJECTS AND METHODS

2.1. Study design

This is a prospective descriptive study conducted at the Department of Emergency Cardiology and Interventional Cardiology, Thong Nhat Hospital, Ho Chi Minh City, from May 2024 to May 2025.

2.2. Study Population

The study included patients aged ≥65 years diagnosed with ACS and CKD who were indicated for PCI.

2.3. Inclusion Criteria

Age ≥65 years.

Diagnosis of ACS, including ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), or unstable angina.

CKD with an eGFR <60 mL/min/1.73 m² for \geq 3 months [6].

Indication for coronary angiography and PCI.

Consent to participate in the study..

Exclusion Criteria:

AKI from other causes at the time of hospital admission.

Patients undergoing chronic dialysis or requiring immediate dialysis.

Allergy to contrast agents.

Hemodynamic instability due to shock or severe infection..

Sample Size and Sampling Method:

A convenience sampling method was used, including all eligible patients during the study period. A total of 225 patients were included in the analysis.

2.4. Study Procedure

patients underwent clinical ΑII evaluations laboratory and tests (creatinine, eGFR, complete blood count, HbA1C, cardiac enzymes, NT-proBNP, etc.), chest X-rays, echocardiography, and electrocardiograms. Renal function was monitored at the following time points: before intervention, within the first 24 hours, days 3–5, and at discharge. CIN prevention was performed using 0.9% NaCl infusion according to the protocol: 3 mL/kg/hour for 1 hour before the intervention and 1-1.5 mL/kg/hour for 4–12 hours afterward [4].

2.5. Variable Definitions

Contrast-Induced Nephropathy (CIN): Defined as an increase in serum creatinine ≥25% or ≥0.5 mg/dL from baseline within 48–120 hours after exposure to contrast agents, excluding other causes [3].

Chronic Kidney Disease (CKD): Defined as eGFR <60 mL/min/1.73 m 2 for \geq 3 months according to KDIGO classification [5].

eGFR (Estimated Glomerular Filtration Rate): Estimated using the CKD-EPI formula, which is officially recommended for assessing and classifying CKD stages [5].

Anemia: Defined as hemoglobin <13 g/dL in men and <12 g/dL in women, per WHO criteria.

Chronic Heart Failure: Defined as reduced left ventricular function (EF <40%) or a history of long-term heart failure treatment.

Hypotension: Defined as systolic blood pressure <90 mmHg at the time of hospital admission.

Mehran Score: A risk prediction tool for CIN consisting of 8 factors: hypotension, intra-aortic balloon pump (IABP), heart failure, age > 75, anemia, diabetes, contrast agent volume, and eGFR < 60 mL/min/1.73 m² [3].

2.6. Data Processing and Analysis:

Data were processed using SPSS

version 25.0. Quantitative variables were presented as mean \pm standard deviation and compared using t-tests or Mann–Whitney tests, depending on distribution. Qualitative variables were compared using the Chi-square test. Statistical significance was defined as p < 0.05.

2.7. Ethical Considerations

The study was approved by the Biomedical Ethics Committee of Thong Nhat Hospital under decision number 42/2024/BVTN-HDYÐ. Patient data were kept confidential and used solely for research purposes..

3. RESULTS

A total of 225 patients aged ≥65 years with CKD and ACS were included in the analysis. Among them, 185 patients (82.2%) were in stage 3 CKD, and 40 patients (17.8%) were in stages 4–5 CKD without chronic dialysis. The average age of the study population was 73.3 ± 1.6 years. The proportion of males was higher than females (57.3% vs. 42.7%). The average hospital stay was 7.16 ± 1.9 days.

In terms of comorbidities, the highest prevalence was hypertension (69.8%), followed by dyslipidemia (67.1%), diabetes

Table 1. General Characteristics of the Study Participants.

Đặc điểm	Chung (n=225)	CKD 3 (n=185)	CKD 4-5 (n=40)	р
Age (years) (Mean ± SD)	73.3 ± 1.6	72.2 ± 1.5	78.1 ± 1.9	0.541
Length of hospital stay (days)	7.16 ± 1.9	7.1 ± 1.9	7.2 ± 1.7	0.475
Gender Male, n (%)	129 (57,3)	110 (59,5)	19 (47,5)	- 0,166
Female, n (%)	96 (42,7)	75 (40,5)	21 (52,5)	
BMI (kg/m²) (Mean ± SD)	23,7±1,7	22,9±2,0	24,5±3,1	0,421
Hypertension, n (%)	157 (69,8)	138 (74,6)	19 (47,5)	0,001
Dyslipidemia, n (%)	151 (67,1)	127 (68,6)	24 (60,0)	0,291
Diabetes mellitus, n (%)	88 (39,1)	75 (40,5)	13 (32,5)	0,345
Overweight (>24 kg/m²), n (%)	42 (18,7)	37 (20,0)	5 (12,5)	0,061
Smoking, n (%)	39 (17.3)	27 (14,6)	12 (30,0)	0,020
Chronic anemia, n (%)	30 (13,3)	10 (5,4)	20 (50,0)	<0,001
Chronic heart failure, n (%)	17 (7,6)	9 (4,9)	8 (20,0)	0,001
Chronic coronary artery disease not revascularized, n (%)	91 (40,4)	78 (42,2)	13 (32,5)	0,259

CKD: Chronic Kidney Disease, SD: Standard Deviation, BMI: Body Mass Index.

Table 2. Clinical and Laborate	ory Characteristics	of Study	Participants at	Admission
Characteristic	Overall (n = 225)	CKD 3 (n = 185)	CKD 4-5 (n = 40)	p-value
	Clinical, n (%	6)		
Dyspnea	209 (92,9)	176 (95,1)	33 (82,5)	0,005
Shortness of breath	81 (36,00	51 (27,6)	30 (75,0)	0,001
Blood pressure (BP), Mean ± SD	125±1.4	128,8±7.4	107,2±7,5	0,267
BP >140 mmHg	50 (22,2)	38 (24,0)	12 (30,0)	0,245
BP <90 mmHg	22 (9,8)	7 (3,8)	15 (37,5)	0,001
Heart rate (Mean ± SD)	78,7±1.2	78,6±8.3	78,4±7,6	0,367
Arrhythmia at admission, n (%)	55 (24,4)	40 (21,6)	15 (37,5)	0,034
Sốc tim	15 (6,6)	4 (2,16)	11 (27,5)	0,008
	Laboratory Charac	teristics		
	Creatinin (µm	ol/L)		
At admission	126,3±2,8	106,5±6,4	217,1±6,9	0,003
Day 1-2	135,9±2,2	110,0±4,1	254,8±5,7	0,002
Day 3-5	160,6±2,8	108,4±4,2	402,7±8,4	0,002
At discharge	116,5±2,5	92,7±2,0	227,6±1,1	0,002
Estir	mated GFR (eGFR m	l/ph/1,73 m²)		
At admission	42,3±1,4	47,3±2,1	20,0±3,1	0,007
Day 1-2	40,6±1,2	45,8±3,4	16,9±1,1	0,008
Day 3-5	39,2±1,6	46,6±1,9	16,8±1,1	0,001
At discharge	47.8 ± 1.7	$54,0\pm2,4$	18,8±1,2	0,003
В	lood count on admi	ssion, n(%)		
Red blood cell count (M/μL)	4,1±0,6	4,2±0,5	4,1±0,9	<0,001
Hemoglobin (g/dL)	12,2±0,9	12,5±1,3	11,2±3,0	<0,001
Anemia, n (%)	66 (29,3)	38 (20,5)	28 (70,0)	<0,001

CKD: Chronic Kidney Disease; SD: Standard Deviation; BP: Blood Pressure; EF: Ejection Fraction; eGFR: Estimated Glomerular Filtration Rate; LVEF: Left Ventricular Ejection Fraction

Phân độ suy tim theo sức co bóp cơ tim n (%)

 $7,3 \pm 1,3$

54 (70,1)

101 (54,6)

20 (10,4)

64 (34,6)

 $7,4 \pm 1,4$

69 (73,4)

123 (54,7)

28 (12,4)

74 (32,9)

mellitus (39.1%), BMI ≥24 (18.7%), and smoking (17.3%).

HbA1c (Mean ± SD)

HbA1c > 6.5%, n (%)

LVEF giảm ≤40%

LVEF giảm nhe 41-49%

LVEF bảo tồn ≥ 50%

6.6% (15/225) of patients were admitted with cardiogenic shock. 73.4% of patients had poorly controlled HbA1C at admission (>6.5%), with mean HbA1c of 7.4±1.4. Mean serum creatinine at admission was 126.3±2.8 and continued to increase, peaking on days 3-5 post-intervention at

 160.6 ± 2.8 . Mean eGFR at admission was 42.3 ± 1.4 and at discharge was 47.8 ± 1.7 ml/min/1.73 m² (Table 2).

7,8±1,4

15 (88,2)

22 (55,0)

8 (20,0)

10 (25,0)

Among clinical forms of acute coronary syndrome, unstable angina accounted for the highest proportion at 56.9%, followed by acute STEMI at 24.4%, and acute NSTEMI at 18.7%. Mean Mehran score was 8.75±1.2 points (Table 3).

0,126

0,126

0,207

0,426

0,321

Table 3. Clinical diagnostic characteristics and Mehran scores at admission.

Characteristics	Overall (n=225)	CKD 3 (n=185)	CKD 4-5 (n=40)	p-value
Diagnosis, n (%)				
CAD with HF	55 (24,4)	31 (16,8)	24 (60,0)	0,001
CAD without HF	42 (18,7)	26 (14,1)	16 (40,0)	0,004
DM without CKD	128 (56,9)	109 (58,9)	19 (47,5)	0,278
Mehran Score, Mean ± SD	8,75±1,2	7,31±1,4	15,4±1,7	0,001
Mehran <10 points, n (%)	165 (73,3)	152 (82,1)	13 (32,5)	0,291
Mehran ≥10 points, n (%)	60 (26,7)	24 (13,0)	36 (90,0)	0,345

CKD: chronic kidney disease, AMI: acute myocardial infarction, STEMI: ST-elevation, UA: unstable angina, Mean: average, SD: standard deviation

Table 4. Intervention results and events after coronary angiography/intervention.

Variables	Overall (n=225)	CKD 3 (n=185)	CKD 4-5 (n=40)	р		
Percuta	Percutaneous Coronary Intervention, n (%)					
Emergency	104 (46,2)	85 (45,9)	19 (47,5)	0,858		
Elective	121 (53,8)	100 (54,1)	21 (52,5)	0,768		
Partial revascularization	88 (39,1)	71 (38,4)	17 (42,5)	0,579		
Complete revascularization	111 (49,3)	94 (50,8)	17 (42,5)	0,478		
Successful procedure	221 (98,2)	183 (98,9)	38 (95,0)	0,348		
Balloon angioplasty without stent	4 (1,8)	1 (0,54)	3 (7,5)	0,003		
Repeat intervention	1 (0.4)	1 (0.54)	0 (0.0)			
Contrast volume (ml), Mean ± SD	227,3±11,6	229,7±6,4	215,3±7,9	0,001		
Number of stents, Mean ± SD	2,3±1,2	2,2±1,3	2,5±1,0	0,198		
Coronary Artery Disease Pattern, n (%)						
Three-vessel disease	94 (41,8)	59 (38,8)	35 (47,9)	0,411		
Three-vessel and left main disease	57 (25,3)	37 (24,3)	20 (27,4)	0,513		
Contrast Agent Type						
Omnipaque (iohexol)	183 (81,3)	175 (94,6)	8 (20,0)	- <0,001		
Visipaque (iodixanol)	42 (18,7)	10 (5,4)	32 (80,0)			
Procedure-Related Complications, n (%)						
Acute anemia	12 (5,3)	7 (3,7)	5 (12,5)	0,041		

Contrast-induced acute kidney injury	31 (13,8)	14 (9,2)	17 (23,3)	0,004
Emergency dialysis	2 (0,9)	0 (0,0)	2 (5,0)	0,002
Acute heart failure	19 (8,4)	9 (5,9)	10 (13,7)	0,049
Intra-aortic balloon pump	4 (1,8)	2 (1,08)	2 (5,0)	0,541
Temporary pacemaker during procedure	25 (11,1)	17 (9,1)	8 (20,0)	<0,001
Cardiogenic shock	11 (4,9)	6 (3,2)	5 (12,5)	0,042
Cardiac arrhythmia	29 (12,9)	20 (10,8)	9 (22,5)	0,045
Hospital-acquired infection	13 (5,7)	8 (4,3)	5 (12,5)	0,032
Stroke	0 (0,0)	0 (0,0)	0 (0,0)	
In-hospital mortality	4 (1,8)	1 (0,5)	3 (7,5)	0,003

CKD: chronic kidney disease, Mean: average, SD: standard deviation

98.2% (221/225) of patients underwent successful intervention with TIMI3 flow. There was 1 case requiring re-intervention due to acute stent thrombosis, 4 cases (1.8%) with balloon angioplasty only without stent placement, and 4 cases (1.8%) died after intervention. Average contrast volume per patient was 227.3 ± 8.6 ml, and 13.8% of patients developed CIN after intervention.

4. DISCUSSION

Our study showed that the incidence of contrast-induced acute kidney injury (CIN) in patients ≥65 years old with chronic kidney disease and acute coronary syndrome after percutaneous coronary intervention was 13.8%. This is a relatively high rate, especially considering that this patient group simultaneously carries multiple risk factors such as: advanced age, hemodynamic disorders, baseline kidney function impairment, and complex cardiovascular disease. This equivalent to international studies in highrisk populations such as McCullough (11– 16%), Mehran (22% in very high-risk group) , and confirmed through systematic review by James and colleagues, in which CIN was associated with increased mortality and emergency dialysis rates after coronary angiography.

A notable finding is that CIN incidence in CKD stage 4–5 group was significantly

higher than in CKD stage 3 group. This reflects the role of baseline kidney function as an independent prognostic factor for CIN, which has been mentioned in many documents, including pathophysiological mechanisms such as increased oxidative stress, reduced renal cortical perfusion, and direct toxicity to tubular cells .

Besides baseline eGFR factor, our study also identified other risk factors including Mehran score ≥10, anemia, chronic heart failure, hypotension, and contrast agent volume used. These are factors that have been systematized in recent guidelines on CIN prevention, emphasizing the role of pre-intervention risk assessment and adjusting treatment strategies according to risk level. Particularly, anemia and heart failure contribute to reducing renal tissue oxygen reserves, making kidneys more vulnerable to damage when exposed to contrast agents.

Some recent studies have raised questions about the causal relationship between CIN and adverse clinical events, suggesting that CIN may be a concurrent manifestation of severe illness rather than an independent cause . However, in real clinical context, especially in patients with severe CKD as in this study, CIN is often accompanied by increased emergency dialysis rates, prolonged hospitalization, and mortality — which has been demonstrated in many large retrospective and prospective analyses .

From these findings, risk assessment using Mehran score, limiting contrast agent dosage, optimizing circulating volume, as well as using low-osmolar contrast agents are evidence-based strategies for effective CIN prevention . Additionally, monitoring serum creatinine and eGFR for at least 5 days after PCI should be routinely applied in CKD patients ≥stage 3, especially when additional risk factors are present.

5. CONCLUSION

Contrast-induced acute kidney injury is a clinically significant complication in patients ≥65 years old with chronic kidney disease and acute coronary syndrome after percutaneous coronary intervention. The incidence rate is significantly higher in patients with severe CKD stages. Factors closely related to CIN risk include Mehran score ≥10, anemia, chronic heart failure, hypotension, and high contrast agent dosage. Early identification of risk factors implementation of appropriate preventive measures play a key role in limiting complications and improving clinical outcomes.

REFERENCES

- [1] McCullough PA, et al. Contrast-Induced Acute Kidney Injury. J Am Coll Cardiol. 2016;68(13):1465-1473.
- [2] Solomon R, Dauerman HL. Contrast-induced acute kidney injury. Circulation. 2010;122(23):2451-2455.
- [3] Mehran R, et al. A simple risk score for prediction of contrast-induced nephropathy after PCI. J Am Coll Cardiol. 2004;44(7):1393–1399.
- [4] Ali M, Nakhoul G, et al. Contrast media in patients with kidney disease: An update. Cleve Clin J Med. 2020;87(11):683–694.
- [5] KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl. 2013;3(1):1– 150.
- [6] Silver SA, et al. Prevention of contrast-associated acute kidney injury: Guideline-based review and update. Can J Kidney Health Dis. 2020;7:2054358120902618.
- [7] James MT, et al. Contrast-induced acute kidney injury and risk of adverse clinical outcomes after coronary angiography: a systematic review and meta-analysis. Circ Cardiovasc Interv. 2013;6(1):37–43.
- [8] Seeliger E, et al. Contrast-induced kidney injury: mechanisms, risk factors, and preven-

tion. Eur Heart J. 2012;33(16):2007-2015.

[9] Rudnick MR, et al. The Controversy of Contrast-Induced Nephropathy With Intravenous Contrast: What Is the Risk? Am J Kidney Dis. 2020;75(1):105–113.