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Original Article

Elevated Arterial Lactate as a Predictor of Contrast-Induced Nephropathy following Percutaneous Coronary Intervention

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ABSTRACT

Background: The incidence of contrast-induced nephropathy (CIN) varies from 0% to 24%. Its mechanism is multifactorial and still not yet fully understood.
In our study, we aimed to evaluate the role of elevated arterial lactate as a predictor of CIN following percutaneous coronary intervention (PCI) in diabetic patients with acute coronary syndrome (ACS).

Methods: This prospective study enrolled 200 diabetic patients with ACS who underwent PCI. Patients with hemodynamic instability, renal impairment, or recent contrast exposure were excluded. Arterial lactate was measured on admission and serum creatinine at 0 and 72 hours. CIN was defined as a rise in serum creatinine of at least 0.5 mg/dL or a 25% increase from baseline within 48–72 hours after contrast exposure.

Results: The mean age was 60 ± 10.34 years, 64% were males, and 33% developed CIN (n = 66). Higher arterial lactate was directly proportional to CIN development ($P < 0.001$). Other predictors were the heart failure signs, procedure duration, and contrast volume >100 mL. In multivariate regression, the most significant predictors were preprocedural arterial lactate > 1 mmol/L (OR 3.932, 95% CI 1.765 to 8.759; $P = 0.001$) and procedure duration > 69 minutes (OR 4.180, 95% CI 1.884 to 9.272; $P < 0.001$).

Conclusions: Preprocedural arterial lactate >1 mmol/L, and procedure duration > 69 minutes were the most important predictors of CIN in diabetic patients with ACS who underwent PCI. Other risk factors were heart failure signs and contrast volume > 100 mL. ***(Iranian Heart Journal 2025; 26(2): 23-32)***

KEYWORDS: Contrast-induced nephropathy**,** Arterial lactate, Percutaneous coronary intervention, Acute coronary syndrome

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One of the most dangerous complications following angiographic procedures is contrast-induced nephropathy (CIN), a term used to describe acute renal failure (ARF) after procedures involving contrast agents. [1, 2]

The incidence of CIN after contrast-based procedures varies widely, ranging from 0 to 24%, depending on patient risk factors and characteristics. [3] CIN is typically a reversible and transient form of ARF. Nonetheless, it is associated with higher morbidity and mortality rates, prolonged hospital stays, and

increased healthcare costs. [4] The management of CIN is primarily supportive, involving careful monitoring and replacement of fluids and electrolytes. Nevertheless, in some cases, it may require hemodialysis. As a general rule, the focus of CIN management should be on prevention. [5]
Despite extensive and ongoing research in this field, the pathophysiology of CIN is not yet fully understood. It is multifactorial and involves various mechanisms, including oxidative stress, reduced renal blood flow, vasoconstriction, renal medullary ischemia, and allergic reactions to contrast agents. [6]
Most tissues in the body produce the chemical compound lactic acid. Blood lactate levels typically range between 0.5 and 1 mmol/L. A prolonged increase in lactate levels to between 2 and 4 mmol/L, without the occurrence of metabolic acidosis, is termed "hyperlactatemia." This condition arises when tissue oxygenation and perfusion remain sufficient. Lactic acidosis, on the other hand, is characterized by lactate levels > 4 mmol/L, which disrupts the acid-base balance, leading to metabolic acidosis with a serum pH < 7.35. [7]
Blood lactate levels are frequently used to assess tissue perfusion. Given that hypoperfusion and ischemia are well-established risk factors for CIN, we aimed to investigate the relationship between blood lactate levels and the occurrence of CIN following percutaneous coronary intervention (PCI) in patients with acute coronary syndrome (ACS).

AIM OF THE WORK

To investigate the significance of elevated arterial lactate levels as a predictor of CIN following PCI in diabetic patients presenting with ACS

METHODS

This study was conducted prospectively between September 2023 and February 2024. Patients meeting the following inclusion criteria were enrolled: diagnosis of ACS (ST-elevation myocardial infarction, non–ST-elevation myocardial infarction, or unstable angina), diabetes mellitus (DM), and management with PCI only. Patients with the following criteria were excluded: hemodynamic instability on presentation or before PCI, a history of chronic kidney disease (including hemodialysis or renal transplantation), chronic obstructive pulmonary disease, diabetic coma or ketoacidosis, a history of or active malignancy, recent use of nephrotoxic drugs (including loop diuretics or aminoglycosides) within 14 days, a recent procedure involving contrast (eg, computed tomography or magnetic resonance imaging) within 14 days before presentation, and anaphylaxis to contrast agents.

All patients underwent the following:

1. **Detailed history taking**, including age, duration of DM, DM medications, and history of renal impairment or renal replacement therapy.
2. **Physical examination**, which included weight, height, body surface area (calculated using the Mosteller method), arterial blood pressure measurement, and general and local examination for Killip classification [8] (patients in Killip classes III and IV were excluded):
    - **Killip I**: No signs of heart failure
    - **Killip II**: Fine basal crepitations on chest auscultation, S3 gallop at the apex on cardiac auscultation, or elevated jugular venous pressure
    - **Killip III**: Acute pulmonary edema
    - **Killip IV**: Cardiogenic shock, hypotension (systolic arterial blood pressure < 90 mm Hg), or evidence of hypoperfusion (eg, cold extremities or oliguria).
3. **Twelve-lead surface ECG** on admission and after the intervention

4. **Laboratory tests,** including
   - Arterial lactate: A sample of arterial blood was obtained from the right radial artery, drawn into a capillary tube, and tested using the Gem 3500 machine.
   - **Pre- and postprocedural serum creatinine (Scr) levels:** Scr was measured at 0 and 72 hours using the Cobas 6000 machine. CIN was defined as an increase in serum creatinine ≥ 0.5 mg/dL or a rise of ≥ 25% compared to the baseline within the first 48 to 72 hours after exposure to contrast agents. [9]
   - **Estimated glomerular filtration rate (eGFR):** eGFR was calculated using the Modification of Diet in Renal Disease (MDRD) equation: [186 × Scr (mg/dL) - 1.154] × age - 0.203 × 0.742 (if female). [10]
   - **Baseline Mehran score:** The Mehran score was determined for all patients before the procedure. [11]
   - **Other laboratory tests:** Random blood sugar
5. **Transthoracic echocardiography (TTE):** All patients underwent a full TTE study using a Vivid E95 echocardiography machine and a 3.5 MHz transducer (GE Healthcare) with emphasis on left ventricular ejection fraction.
6. **Coronary angiography and PCI**: All patients underwent PCI by expert interventional cardiologists using the same contrast agent (Omnipaque), and the following data were obtained: total contrast volume (mL), total fluoroscopy time, and total procedure duration.

Statistical Analysis:

Codes, tabulations, and statistical analyses were performed on the data using IBM SPSS Statistics version 18.0, IBM Corp, 2009. For quantitative data that were normally distributed, descriptive statistics were computed as mean ± standard deviation (SD) and the lowest and maximum of the range. For qualitative data, they were calculated as numbers and percentages. The independent *t*-test was applied to data that were normally distributed for 2 independent groups. The ANOVA test with a post hoc Tukey test was applied for data that were normally distributed across more than 2 separate groups. Additional inferential analyses were conducted for the quantitative variables, and the Shapiro-Wilk test was used to test for normality. Inferential analysis of independent variables was carried out when evaluating qualitative data, employing the Fisher exact test for variables with small expected numbers and the post hoc Bonferroni test for discrepancies between proportions. Moreover, Logistic regression was applied to find independent factors driving conduction anomalies. The performance of various tests was assessed, and certain groups were distinguished from one another using the receiver operating characteristic (ROC) curve. A *P*-value < 0.050 was selected as the significance level.

Research Ethics

This study was conducted in compliance with the rules, regulations, and policies of the Faculty of Medicine at Ain Shams University. Approval was granted on March 21, 2023, with the ethics committee's reference number: FMASU MS 492/2023. Written informed consent was obtained from all patients or their designated representatives before the initiation of any study procedures, in accordance with the WMA Declaration of Helsinki.

RESULTS

The study included 200 patients with a mean age of 60 ± 10.34 years; 64% were male, and 12% exhibited symptoms and signs of congestive heart failure (CHF) limited to

Killip classes I and II. Table 1 provides additional demographic and clinical data.

Regarding periprocedural data, the average serum creatinine level was 0.9 ± 0.27 mg/dL, the Mehran score ranged from 3 to 17, and the contrast volume used ranged from 50 to 300 mL, with an average of 100 mL among the studied patients. CIN occurred in 33% of patients (n = 66). Table 2 displays additional periprocedural data.

Table 1. Demographic and clinical data of the studied patients

Parameters		Total No. = 200
Age, y	Mean ± SD Range	60.08 ± 10.34 33 – 82
Sex	Female Male	72 (36%) 128 (64%)
Height, cm	Mean ± SD Range	1.71 ± 0.08 1.5 – 1.9
BMI	Mean ± SD Range	27.7 ± 2.1 23 – 40
CHF signs and symptoms	No Yes	176 (88%) 24 (12%)
Hypertension	No Yes	51 (25.5%) 149 (74.5%)
Systolic blood pressure, mm Hg	Mean ± SD Range	132.88 ± 22.94 90 – 200
Diastolic blood pressure, mm Hg	Mean ± SD Range	79.7 ± 12.83 50 – 120
Heart rate, bpm	Mean ± SD Range	83.26 ± 14.3 50 – 130

BMI: body mass index, CHF: congestive heart failure

Table 2. Periprocedural parameters among the studied patients

Parameters		Total No. = 200
Preprocedural random blood sugar	Median (IQR)	200 (146 – 261)
	Range	80 – 500
Preprocedural arterial lactate	Median (IQR)	1 (0.8 – 2)
	Range	0.2 – 7.9
Preprocedural serum creatinine	Mean ± SD	0.9 ± 0.27
	Range	0.4 – 1.6
Preprocedural GFR	Median (IQR)	83 (64.2 – 103.6)
	Range	38.9 – 178.2
Preprocedural LVEF	Mean ± SD	50.44 ± 12.67
	Range	20 – 78
Mehran score	Median (IQR) Range	4 (3 – 4) 3 – 17
Postprocedural creatinine	Mean ± SD Range	1.15 ± 0.66 0.3 – 3.9
Postprocedural GFR	Median (IQR) Range	73 (48.4 – 97.8) 11.9 – 298.2
CIN	Non-CIN CIN	134 (67%) 66 (33%)
Whole procedure duration, min	Mean ± SD Range	66.95 ± 20.37 30 – 150
Fluoroscopy time, min	Median (IQR) Range	20 (20 – 30) 10 – 70
Contrast volume	Mean ± SD Range	100 (80 – 100) 50 – 300

GFR: glomerular filtration rate, LVEF: left ventricular ejection fraction, CIN: contrast-induced nephropathy

We investigated the relationship between the incidence of CIN and demographic and clinical data. There was a statistically significant relationship between age and CIN incidence (P = 0.035) and a highly significant relationship between the presence of signs and symptoms of CHF and CIN (P < 0.001), as shown in Table 3.

We also studied the relationship between CIN incidence and periprocedural data. There was a statistically significant relationship between preprocedural arterial lactate (P < 0.001), the Mehran score (P < 0.001), whole procedure duration (P < 0.001), fluoroscopy time (P < 0.001), and total contrast volume (P < 0.001) used, as shown in Table 4.

To determine the cut-off level of arterial lactate that may predict the occurrence of CIN, we utilized the ROC curve. The area under the curve with a cut-off value > 1 mmol/L was 0.740. As shown in Figure 1, applying this cut-off value resulted in an overall sensitivity of 75.76%, specificity of 63.43%, positive predictive value of 50.5%, and negative predictive value of 84%. We performed a multivariate logistics regression analysis, addressing all statistically significant risk factors for CIN among the studied patients. Our findings revealed that preprocedural arterial lactate > 1 mmol/L and whole procedure duration > 69 minutes were the most statistically significant risk factors for CIN, with odds ratios (ORs) of 3.932 (95% CI 1.765 to 8.759; P = 0.001) and 4.180 (95% CI 1.884 to 9.272; P < 0.001), respectively (Table 5).

Table 3. The relationship between the incidence of CIN and the demographic characteristics of the studied patients

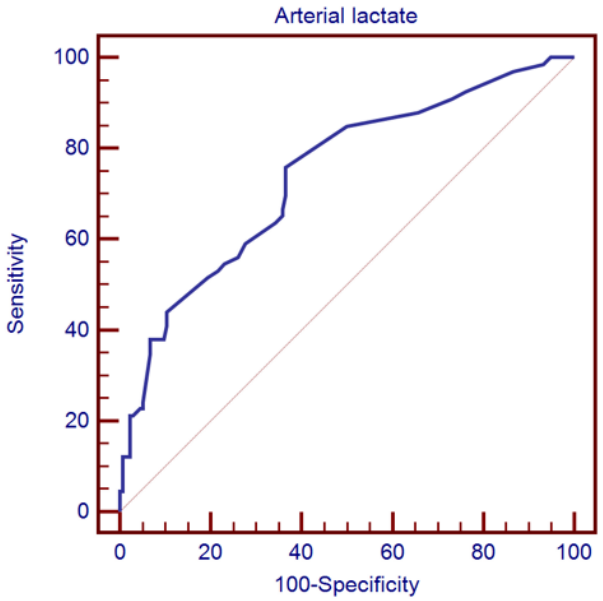
Parameters		Non-CIN No. = 134	CIN No. = 66	Test Value	*P*	Sig.
Age, y	Mean ± SD	59 ± 10.85	62.27 ± 8.91	-2.122•	0.035	S
	Range	33 – 82	38 – 77			
Sex	Female	47 (35.1%)	25 (37.9%)	0.151*	0.698	NS
	Male	87 (64.9%)	41 (62.1%)			
Height, cm	Mean ± SD	1.71 ± 0.07	1.7 ± 0.09	0.671•	0.503	NS
	Range	1.55 – 1.87	1.5 – 1.9			
BMI	Mean ± SD	27.62 ± 1.71	27.88 ± 2.73	-0.839•	0.402	NS
	Range	24 – 32	23 – 40			
CHF signs and symptoms	No	130 (97.0%)	46 (69.7%)	31.250*	<0.001	HS
	Yes	4 (3.0%)	20 (30.3%)			
Hypertension	No	34 (25.4%)	17 (25.8%)	0.003*	0.953	NS
	Yes	100 (74.6%)	49 (74.2%)			
Systolic blood pressure, mm Hg	Mean ± SD	131.42 ± 21.76	135.83 ± 25.08	-1.282•	0.201	NS
	Range	90 – 200	90 – 200			
Diastolic blood pressure, mm Hg	Mean ± SD	79.33 ± 12.58	80.45 ± 13.41	-0.583•	0.561	NS
	Range	50 – 120	50 – 110			
Heart rate, bpm	Mean ± SD	82.55 ± 13.97	84.68 ± 14.96	-0.990•	0.323	NS
	Range	50 – 120	60 – 130			

P > 0.05: (NS) nonsignificant, *P* < 0.05: (S) significant, *P* < 0.01: (HS) highly significant
*: χ^2 test; •: independent *t*-test SD,
CIN: contrast-induced nephropathy

Table 4. The relationship between the incidence of CIN and periprocedural data

		Non-CIN No. = 134	CIN No. = 66	Test Value	*P*	Sig.
Preprocedural random blood sugar	Median (IQR)	200 (146 – 261)	200 (150 – 261)	-0.372≠	0.710	NS
	Range	80 – 500	88 – 475			
Preprocedural arterial lactate	Median (IQR)	0.95 (0.7 – 1.7)	2 (1.1 – 3)	-5.524≠	<0.001	HS
	Range	0.2 – 5.7	0.4 – 7.9			
Preprocedural serum creatinine	Mean ± SD	0.88 ± 0.27	0.95 ± 0.28	-1.939•	0.054	NS
	Range	0.4 – 1.6	0.5 – 1.5			
Preprocedural GFR	Median (IQR)	85.45 (68.3 – 110.2)	77.05 (56 – 94.4)	-2.298≠	0.022	S
	Range	38.9 – 178.2	40.6 – 163			
Preprocedural LVEF	Mean ± SD	52.26 ± 12.38	46.74 ± 12.54	2.951•	0.004	HS
	Range	25 – 78	20 – 76			
Mehran score	Median (IQR)	4(3 – 4)	4(4 – 9)	-6.254≠	<0.001	HS
	Range	3 – 13	3 – 17			
Whole procedure duration, min	Mean ± SD	60.78 ± 15.27	79.47 ± 23.59	-6.747•	<0.001	HS
	Range	30 – 120	30 – 150			
Fluoroscopy time, min	Median (IQR)	20 (18 – 25)	30 (20 – 30)	-3.628≠	<0.001	HS
	Range	10 – 50	10 – 70			
Contrast volume	Mean ± SD	92.8 ± 27.51	124.85 ± 50.94	-5.779•	<0.001	HS
	Range	50 – 200	50 – 300			

P > 0.05: (NS) nonsignificant, *P* < 0.05: (S) significant, *P* < 0.01: (HS) highly significant
•: independent *t*-test; ≠: Mann-Whitney test
CIN: contrast-induced nephropathy, LVEF: left ventricular ejection fraction



Cut off point	AUC	Sensitivity	Specificity	+PV	-PV
>1	0.740	75.76	63.43	50.5	84.2
AUC (AreaUnder Curve), +PV (Positive predictive value), - PV (Negative Predective Value)					

Figure 1. The receiver operating characteristic (ROC) curve assesses arterial lactate as a predictor of CIN among the studied patients.
CIN: contrast-induced nephropathy

Table 5. Univariate and multivariate logistics regression analysis to assess the risk factors for CIN among the studied patients

	Univariate				Multivariate (Backward: Wald)			
	P	OR	95% CI for OR		*P*	OR	95% CI for OR	
			Lower	Upper			Lower	Upper
Age > 64 y	<0.001	3.146	1.701	5.819	0.079	1.998	0.923	4.327
CHF signs and symptoms	<0.001	14.130	4.588	43.522	0.011	5.321	1.479	19.150
Preprocedural arterial lactate >1	<0.001	5.421	2.791	10.527	0.001	3.932	1.765	8.759
Preprocedural GFR ≤ 84.5	0.003	2.592	1.387	4.843	–	–	–	–
Preprocedural LVEF <= 52	<0.001	3.107	1.660	5.815	–	–	–	–
Mehran score >3	<0.001	6.243	2.768	14.083	–	–	–	–
Whole procedure duration >69, min	<0.001	6.645	3.467	12.735	<0.001	4.180	1.884	9.272
Fluoroscopy time >28, min	<0.001	4.022	2.126	7.610	–	–	–	–
Contrast volume >100	<0.001	8.060	3.579	18.150	0.032	3.116	1.105	8.788

P > 0.05: (NS) nonsignificant, *P* < 0.05: (S) significant, *P* < 0.01: (HS) highly significant
•: independent *t*-test; ≠: Mann-Whitney test
GFR: glomerular filtration rate, LVEF: left ventricular ejection fraction, CHF: congestive heart failure

DISCUSSION

Numerous studies have demonstrated that the development of CIN is associated with significant impacts on clinical practice, including extended hospital stays and increased hospital costs, [12,13] Serum lactate is frequently employed as a diagnostic and prognostic tool in critical care settings due to its recognized role as an indicator of tissue perfusion. Blood lactate measurements at specific time points have been shown to affect prognosis, with the duration of elevated blood lactate levels correlating with increased morbidity and mortality. [14] In a relevant study, Zhang and Ni [15] found that elevated blood lactate levels in individuals who underwent cardiopulmonary bypass might be associated with the development of ARF following heart surgery. Gatien et al [16] demonstrated that venous blood lactate levels could be highly sensitive in diagnosing acute myocardial infarction, particularly when anginal pain persists for over 2 hours. Additionally, Vermeulen et al [17] found that higher blood lactate levels were an independent predictor of 30-day mortality and overall worse prognosis following PCI. In our study, we focused on examining the predictive value of preprocedural arterial lactate levels for CIN in diabetic patients diagnosed with ACS who underwent PCI. We found that higher arterial lactate levels were independently correlated with an increased risk of CIN. Additionally, we identified a lactate cut-off value > 1 mmol/L as a significant predictor of CIN occurrence. Our cut-off was low compared with the findings of Gaoliang et al, [18] who reported a cut-off value ≥ 3.02 mmol/L, primarily because we excluded patients with conditions known to elevate lactate levels, such as hemodynamically unstable patients, those with cardiogenic shock, acute pulmonary edema, and diabetic ketoacidosis. In contrast to Gaoliang and colleagues, our results indicated that 33% of 200 patients developed CIN, compared to their finding of 22% out of 280 patients. Our study aligns with the findings of Jun-Qing Yang et al, [19] who investigated the association between elevated preprocedural blood lactate levels and the incidence of CIN in patients diagnosed with ST-segment-elevation myocardial infarction (STEMI) who underwent primary PCI. They discovered that arterial lactate levels > 2 mmol/L were a strong predictor of CIN development following PCI (OR 3.77, 95% CI 1.77 to 7.99; *P* = 0.001). It is important to note that their study excluded hemodynamically unstable patients as well. Furthermore, Göksel Güven et al [20] also

discovered that elevated admission lactate levels > 2.8 mmol/L were associated with an increased risk of CIN, and patients with diabetes were more likely to have high lactate concentrations. In another study, Vermeulen et al [17] reported that lactate levels > 1.8 mmol/L on admission were predictive of worse outcomes and acute mortality in patients with STEMI.

We found a statistically significant positive correlation between the occurrence of CIN and age. Additionally, we observed no statistically significant correlation between body mass index (BMI) or sex distribution and the development of CIN. In contrast, Gaoliang et al [18] demonstrated a statistically significant relationship between the occurrence of CIN and both height and BMI. This discrepancy may be attributed to the fact that most of our patients had a similar range of BMI. In our study, preprocedural serum creatinine levels were not statistically significant, whereas they were significant in the study by Gaoliang and colleagues. This difference may be due to our exclusion of patients with impaired renal function, resulting in serum creatinine levels within the normal range for our cohort. Both studies found preprocedural eGFR and contrast volume to be statistically significant. Our study also found that a procedure duration > 69 minutes was a highly significant predictor of CIN following PCI. This might be due to the possibility that longer procedures are often associated with more complex coronary artery disease and higher contrast volume usage. Notably, our research is the first to establish a relationship between procedure duration and CIN incidence. Still, numerous studies have previously confirmed the correlation between contrast volume used [21-24] and CAD complexity [25,26] in relation to CIN risk.

CONCLUSIONS

Elevated preprocedural arterial lactate (> 1 mmol/L) and procedure duration (> 69 min) were the most significant predictors of CIN development in diabetic patients with ACS undergoing PCI. Other predictors included heart failure signs and symptoms and contrast volume > 100 mL.

Declarations

Ethics Committee Approval and Informed Consent

This study was conducted in compliance with the rules, regulations, and policies of the Faculty of Medicine at Ain Shams University. Approval was granted on March 21, 2023, with the ethics committee's reference number: FMASU MS 492/2023. Written informed consent was obtained from each patient or their authorized representative before initiating any study procedures, in accordance with the WMA Declaration of Helsinki.

Availability of Data and Materials

Data supporting the study results are available upon request.

Conflict of Interest

All authors declare no conflicts of interest.

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Not applicable.

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