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Relationship between Arterial Lactate and Contrast-Induced Nephropathy (CIN) In Primary Percutaneous Coronary Intervention (PCI)

Vishvajit Wakade[1]*, Amitesh Nagarwal[2], Vinod Kr. Poonia[3], Sheshkaran Singh Charan[1], Vishal Gaurab[1], Manna Bhattacharjee[1]

[1]Senior Resident, Department of Cardiology, NIMS University, Jaipur, India
[2]Professor, Department of Cardiology, NIMS University, Jaipur, India
[3]Associate Professor, Department of Cardiology, NIMS University, Jaipur, India

***Address for Correspondence:** Dr. Vishvajit Wakade, Senior Resident, Department of Cardiology, NIMS University, Jaipur, India
E-mail: wakadevishvajit@gmail.com

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ABSTRACT

Background: Contrast-induced nephropathy (CIN), also called contrast-induced acute kidney injury (CI-AKI), is an indicator for renal failure, morbidity, and mortality after PCI. This affects the 13.3 million patients per year, with 1.7 million deaths. The pathogenesis includes oxidative stress, high levels of ROS, cytotoxicity, and renal hypoxia.

Methods: This is a prospective observational study conducted over 18 months in a tertiary care centre in Jaipur, which has assessed the association between pre-procedural arterial lactate and CIN among STEMI patients who underwent PCI. Different criteria and techniques have been used for data collection, followed by standard procedures for statistical analysis.

Results: The study was conducted among 110 patients, with males being much more predominant than females, and the age group 61 to 70 years was the most common. At baseline, CPKMB values ranged from 0.74 to 400, with a mean of 63.86±100.2, indicating high variability. Haemoglobin levels ranged from 6.8 to 16.5 g/dl (mean: 13.17±2.04), and HbA1c levels ranged from 4.2% to 11.1% (mean: 6.1±1.2%). LVEF values varied from 20% to 55% (mean: 39.77±8.66%). Post-intervention, creatinine levels slightly increased (mean: 0.914±0.331). At a lactate cut-off of 1.5 mmol/L, the sensitivity was 92%, accurately identifying 92% of CIN cases. Specificity was 62.4%, correctly identifying 62.4% of non-CIN cases.

Conclusion: The study concluded that Arterial lactate levels before primary percutaneous coronary intervention (PCI) are a significant predictor of contrast-induced nephropathy (CIN).

Key-words: Arterial Lactate, Contrast Nephropathy (CIN), Primary Percutaneous Coronary Intervention (PCI), STEMI, Risk Stratification

INTRODUCTION

Contrast-induced nephropathy (CIN), also referred to as CI-AKI, is a cause of renal failure, a crucial risk factor for mortality, and a cost-effective health care issue [1,2]. Patients who have undergone PCI are highly associated with the risk of CIN [3,4].

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Early diagnosis of CIN has improved with the development of accurate therapeutic strategies, including hydration and statins, among PCI risk populations [5-7]. About 13.3 million patients have been diagnosed with the condition per year, and 1.7 million deaths is associated with the CI-AKI per year [8].

The CI-AKI risk has been observed more in elderly patients and among those who have been diagnosed with renal disorders, diabetes, or other negative conditions [9]. The condition is combined with CKD and accounts for 40%. CI-AKI is also linked with severe kidney failure and delay in hospitalization, as 13% of the hospitalized patients have undergone dialysis and the rate of mortality is about 7 to 31% [10]. About one-third of

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the patients have been observed with CKD in 2 to 5 years after recovering from the AKI condition [11].

The condition is linked with some adverse medical conditions like renal failure and severe cardiovascular disorders. The data suggest that about 0.06% of all patients have received renal replacement therapy due to renal impairment [12,13]. 25 to 30% of the CI-AKI patients have undergone severe renal failure, which increases the hospital stay by about 2 to 10 times [14,15]. The pathogenesis of CI-AKI has been attributed to direct and indirect effects and the formation of reactive oxygen species (ROS) [16].

In the case of direct effects, the nephrons' cytotoxicity has been triggered by the iodinated contrast medium, resulting in mitochondrial impairment, apoptosis, pyroptosis, necrosis, and inflammation in the interstitium. In the case of indirect effects, iodine contrast medium causes alterations in the haemodynamics of the renal region, leading to contraction of the renal blood vessels, along with intramedullary ischemia and a hypoxic condition. These medium result in a high production of ROS, reducing antioxidant enzymatic activity, which enhances oxidative stress and inflammation, thus causing renal impairment [17]. The hypoxic condition of the renal medulla can increase ROS production, resulting in oxidative stress in the mitochondria and mitochondrial impairment [18].

For CIN prevention and management, risk stratification is a crucial step. One study demonstrated that 8 variables extracted from the Mehran Risk Score (MRS), which is used for the predictive analysis of CIN in elective PCI [19], perform better in emergent PCI [20]. There is a systematic review study that presents various models for prediction, including both internal and external values for discrimination in CIN [21]. Also, some studies have observed that neutrophil gelatinase-associated lipocalin and Cystatin C are crucial and significant biomarkers for predicting CIN [22,23]. Lactic acid is formed during anaerobic glycolysis in the absence of oxygen, and its level indicates the status of tissue perfusion. Renal blood flow is impaired, and hypoxia in the renal region is a crucial factor in CIN pathophysiology. Contrast media (CM) is toxic to the vasoconstriction of the renal artery and results in ischemic conditions in the kidney [24].

MATERIALS AND METHODS

Research design- The study is a prospective observational study that helps evaluate the association between pre-procedural arterial lactate levels and CIN among patients undergoing PCI. The study was conducted in the Department of Cardiology, National Institute of Medical Sciences & Research, Jaipur, Rajasthan, in a Tertiary Care Hospital. The hospital is equipped with cardiac catheterization labs, which support cardiovascular interventions. The study was conducted for 18 months, from July 2023 to December 2024. Patient involvement has been done based on various clinical parameters, regular follow-ups, laboratory evaluations, and data analysis. The study patients have been selected based on the visitors to the ED for the acute chest pain and those who were diagnosed with STEMI. It was based on electrocardiographic assessment, with the ST segment≥2 mm in the precordial leads or a new-onset left bundle branch block (LBBB). Those patients who had undergone primary PCI.

Inclusion criteria

- The patients were those who were ≥18 years old, both male and female.
- The patients who were diagnosed with chest pain for 12 to 48 hours and were observed with the ST segment rise of more than 2 mm, for the 2 contiguous precordial ECGs.
- Proper written and verbal consent from patients has been included in the study.

Exclusion criteria

- If any patients have been diagnosed with any anaerobic conditions like cardiopulmonary resuscitation, septicemia, or any shock, they have been excluded.
- Any patients with no pre-procedural arterial lactate values were not considered for the study.
- Any renal disease or any undergoing dialysis condition is not considered for the study.
- Severe diabetic condition with metformin is not allowed.
- The COPD patients have not considered for the study.

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Sample Size

The sample size was calculated using the formula:

Z 2 α/2 × p × (1-p)/d2 = (1.96)2×0.207×(1-0.207)/(0.08)2 = 99 Samples

Zα/2: inverse probability of the normal distribution at 95% confidence interval.

p: Proportion rate of CIN developed

d: Margin of error (8% considered)

Sampling Technique- A purposive sampling technique was used.

Procedure - Participants were selected based on the ED inclusion criteria and transitioned to the cardiac catheterization laboratory. The primary PCI was performed by expert cardiologists using the femoral or radial approach, with standard hardware such as catheters, guidewires, balloon catheters, aspiration catheters, and stents. Lactate levels were measured during the presentation in the hospital or before PCI. The arterial blood is taken from the femoral or radial artery with the help of heparinized Syringes. Lactate levels are evaluated using point-of-care blood gas analyzers. All participants have been administered aspirin 300 mg, clopidogrel 300 mg/Ticagrelor 180 mg before PCI. Iodixanol & Iohexol types of contrast media have been used. The post-PCI creatinine levels were measured after 72 hours to detect CIN. CIN is the high level of creatinine in blood serum, ≥0.5 mg/dl or ≥25% within 48-72 hours.

Statistical analysis- Data were entered in Microsoft Excel and analyzed using SPSS. Continuous variables were tested for normality (Shapiro–Wilk) and presented as mean±SD or median. Categorical variables were expressed as frequencies and percentages. Group comparisons used Student's t-test or Mann–Whitney U test, and Chi-square or Fisher's exact test. Pearson's correlation assessed the association between arterial lactate and CIN, and multivariable logistic regression identified CIN predictors. Statistical significance was set at $p<0.05$.

Ethical consideration- Ethical approval was obtained from the Institutional Ethics Committee, NIMS, Jaipur. Written informed consent was secured from participants or their representatives. Patient data were anonymized to ensure confidentiality, and the study adhered to the Declaration of Helsinki and ICMR guidelines.

RESULTS

Table 1 shows the distribution of age and gender among the 110 participants. The largest group is aged 61-70 years, comprising 45.45% of the sample, followed by the 51-60 age range at 19.09%. Younger age groups, specifically those aged ≤40 years (5.45%) and 41-50 years (16.36%), make up a smaller portion. Notably, no participants were in the 71-75 age range. In terms of gender, the study had a male-dominated sample, with 75.45% males and 24.55% females. The age distribution reflects a predominantly older patient population.

Table 1: The representation of the age ranges and the gender, along with their percentages

Age Interval	n = 110	In %
≤ 40	6	5.45
41 - 50	18	16.36
51 - 60	21	19.09
61 - 70	50	45.45
71 - 75	0	0
> 75	15	13.64
Gender		
Male	83	75.45
Female	27	24.55

Table 2 presents a comparative study of creatinine levels at the pre- and post-interventional stages among 110 patients. The majority (50.91%) have consistent creatinine levels of 0.76–1.00 mg/dl. There is a slight decline in the 0.51–0.75 mg/dl range, but rises in the 1.01–1.25 mg/dl and >1.25 mg/dl ranges. Thus, creatinine levels in the stable group showed few changes during the intervention.

Table 2: The distribution of the level of creatinine in the pre- and post-interventional stage

Creatinine (mg/dl)	Pre Intervention		Post Intervention	
	n = 110	In %	n = 110	In %
0.25 - 0.50	5	4.55	5	4.55
0.51 - 0.75	29	26.36	27	24.55
0.76 - 1.00	56	50.91	56	50.91
1.01 - 1.25	11	10	12	10.91
> 1.25	9	8.18	10	9.09

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Table 3 presents descriptive statistics of clinical variables at baseline and post-intervention. CPK-MB ranged from 0.74 to 400, with a median of 23.55 (IQR: 5.97–69.1) and mean±SD of 63.86±100.2, showing considerable variability. Haemoglobin (Hb) varied from 6.8 to 16.5 g/dl, median 13.25 (IQR: 12.4–14.2), mean 13.17±2.04, indicating moderate spread. HbA1c ranged 4.2–11.1%, median 5.8% (IQR: 5.6–6.4), mean 6.1±1.2%, reflecting relatively controlled glucose levels. LVEF ranged 20–55%, median 40% (IQR: 35–45), mean 39.77±8.66%, indicating variable cardiac function. Lactate levels varied 0.5–15 mmol/L, median 1.5 (IQR: 1.1–1.9), mean 1.88±2.02, showing low but diverse concentrations. Creatinine at baseline ranged 0.5–1.5 g/dl, median 0.9 (IQR: 0.6–1.0), mean 0.862±0.243, and post-intervention 0.45–2.04 g/dl, median 0.9 (IQR: 0.7–1.0), mean 0.914±0.331, indicating slight post-intervention changes.

Table 3: Descriptive Statistics of Key Variables (CPKMB, Hb, HbA1c, Lactate, Creatinine) at Baseline and After Intervention

Variables		Minimum	Maximum	Median (IQR)	Mean±SD
CPKMB		0.74	400	23.55 (5.97-69.1)	63.86±100.2
Hb (g/dl)		6.8	16.5	13.25 (12.4-14.2)	13.17±2.04
HbA1c (%)		4.2	11.1	5.8 (5.6-6.4)	6.1±1.2
LVEF (%)		20	55	40 (35-45)	39.77±8.66
Lactate (mmol/L)		0.5	15	1.5 (1.1-1.9)	1.88±2.02
Creatinine (mg/dl)	At Baseline	0.5	1.5	0.9 (0.6-1)	0.862±0.24
	After Intervention	0.45	2.04	0.9 (0.7-1)	0.914±0.33

Fig. 1 shows the distribution of creatinine levels in patients before and after an intervention. The x-axis represents creatinine level categories, and the y-axis shows the number of patients in each category. Before the intervention, most patients had creatinine levels between 0.76 and 1, with 56 patients in this range. After the intervention, the number of patients in the 0.76–1 range remained high (56), but more patients shifted to lower creatinine levels (0.25–0.50 and 0.51–0.75). Overall, the intervention seems to have led to a shift towards lower creatinine levels in patients.

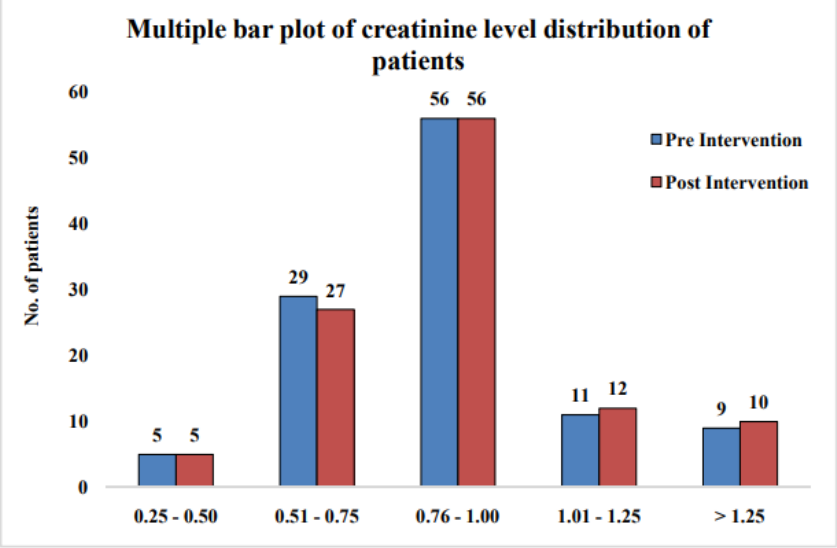

Fig. 1: Plot showing creatinine level distribution of patients

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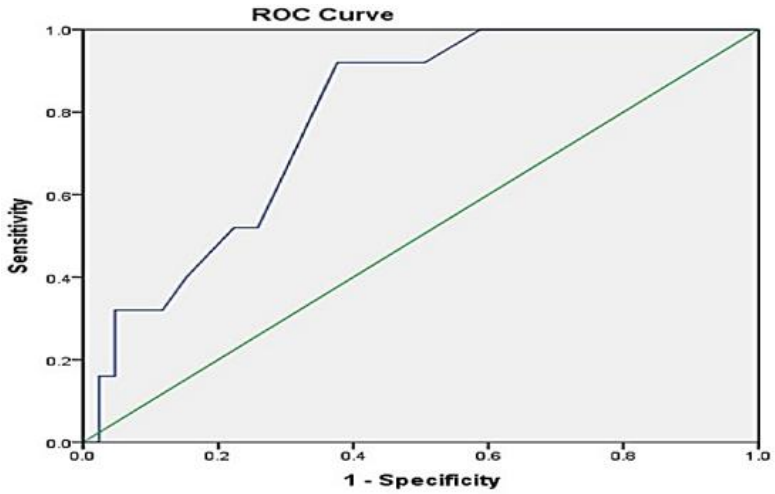
Table 4 shows the relationship between lactate levels and various other clinical parameters. Age and contrast volume are not associated with a rise in lactate levels. But diabetes mellitus was associated with patients with a high lactate level ≥1.5 mmol/L. Also, congestive heart failure (CHF) is associated with high levels of lactate in CHF patients. Anaemia is associated with elevated lactate levels.

Table 4: The value of the level of lactate according to the variables considered.

Variables		Lactate (< 1.5 mmol/L)	Lactate (≥ 1.50 mmol/L)	Chi-Square	p-value	Significance
Age	< 75 Yrs.	49	46	0.69	0.40	Not Significant
	≥ 75 Yrs.	6	9			
Diabetes Miletus	Yes	5	17	8.18	0.004	Significant
	No	50	38			
Contrast Volume	≤ 150 ml	27	18	3.05	0.08	Not Significant
	> 150 ml	28	37			
CHF	Yes	2	13	9.34	0.002	Significant
	No	53	42			
Anemia	Yes	12	22	4.25	0.03	Significant
	No	43	33			

Fig. 2 shows the Receiver Operating Characteristic (ROC) curve for the association of pre-procedural arterial lactate levels in predicting CIN. The ROC curve demonstrates the trade-off between sensitivity and specificity at various cut-off points. The AUC (Area Under the Curve) is 0.784, suggesting a good model fit for predicting CIN. The sensitivity at the cut-off value of 1.5 is 92%, meaning the test correctly identifies 92% of CIN cases. Specificity is 62.4%, indicating that 62.4% of non-CIN cases are correctly identified. The 95% confidence interval for the AUC ranges from 0.697 to 0.871, reinforcing the model's reliability.



AUC	Std. Error	95% CI	Cut off Value	Sensitivity	Specificity
0.784	0.044	0.697 - 0.871	1.5	92%	62.40%

Fig. 2: Association of pre-procedural arterial lactate level in the prediction of CIN and its ROC

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DISCUSSION

The study compares creatinine levels before and after an intervention in 110 patients and shows that most patients maintained stable creatinine levels, with minimal changes across categories. A slight decrease in creatinine levels was observed post-intervention, but the overall impact was limited. Descriptive statistics revealed considerable variability in clinical parameters like CPKMB, haemoglobin, HbA1c, LVEF, and lactate levels, with slight post-intervention changes in creatinine. Lactate levels were significantly associated with diabetes, congestive heart failure, and anaemia, but not with age or contrast volume. The ROC curve analysis indicated that pre-procedural arterial lactate levels had a strong predictive value for CIN, with high sensitivity and moderate specificity.

Elevated levels of lactate have been linked with the high mortality rate in hospitals and the long duration of hospital stay. Arterial lactate is a readily available biomarker that effectively stratifies CIN in the PCI risk population [25]. The study found an association between lactate and CI-AKI among 843 AMI patients who underwent emergent PCI. The CI-AKI patients had a high lactate level, and those with lactate ≥2.5 mmol/L were more common. Multivariate analysis has been performed, which indicates that a high lactate level is crucial for CI-AKI prediction, with an odds ratio of 2.6. The high level of lactate has been associated with the high mortality rate in hospitals with major cardiovascular conditions. The paper reveals that lactate concentration in blood serum is a major diagnostic biomarker for identifying both renal and clinical improvements among AMI patients undergoing PCI [26].

Lactate and its clearance have been crucial for predicting AKI in STEMI patients who have undergone PCI. The paper reveals that lactate levels >2.0 mmol/L are associated with a high level of CI-AKI and a doubling rate. The low level of lactate clearance is between 12 and 24 hours, which is associated with renal recovery and an increase in short-term mortality rates. Many studies have been conducted with the odds ratios of 1 to 3 in case of elevated lactate levels, which is a risk for AKI patients. The study has highlighted that the constant level of lactate and the clearance rate are crucial for prognosis, suggesting that lactate is the major biomarker for stratifying risks [27].

A study by Masoomi *et al.* evaluated the prevalence and risk factors associated with CIN among patients who have undergone PCI. Out of 412 patients, the overall CIN was 12.9%, mainly observed in cases of old patients and among patients who have been diagnosed with diabetes and renal abnormalities. CIN patients have been observed with a high duration of stay in the hospital and a high mortality rate. In the multivariate analysis, diabetes, hypertension, contrast volume, and EGFR were identified as important predictors. The study has identified the CIN and the major complications associated with PCI, leading to early identification and significant prevention among the risk populations [28].

The studies that have resulted from the review analysis of CI-AKI have explained the mechanisms and provided significant preventive measures. 7–15% of patients who have undergone PCI have been observed to develop CI-AKI, whereas 20-30% were observed in risk populations, such as diabetes or CKD, which results in renal vasoconstriction, oxidative stress, and tubular toxicity towards the CM. The patient mortality rate is 2 to 5 times higher than that of the individual without any renal injury. The paper suggests that proper hydration, limiting the contrast volume, and using low-osmolar or iso-osmolar agents. The study highlights that CI-AKI is a crucial complication that requires immediate diagnostic intervention, while maintaining the risks associated with it and its specific prevention [29].

LIMITATION

The limitations of this study include its observational design, which may introduce bias and limit the ability to establish causal relationships. Additionally, the study was conducted at a single tertiary care centre, which may affect the generalizability of the findings to other populations. The sample size, although calculated, remains relatively small, which may affect statistical power. Moreover, the study does not account for other potential confounding factors that could affect the development of CIN.

CONCLUSIONS

Arterial lactate levels before primary PCI are a significant predictor of CIN. A lactate level greater than 1.5 mmol/L was associated with a higher incidence of CIN, and lactate levels demonstrated high sensitivity and moderate specificity for predicting CIN. This study

highlights the importance of pre-procedural arterial lactate as a reliable predictor for CIN in patients undergoing primary PCI. Elevated lactate levels (≥1.5 mmol/L) were strongly associated with CIN development, and multivariate analyses confirmed lactate's role as a significant predictor. Lactate levels showed good sensitivity (92%) and moderate specificity (62.4%) for identifying CIN. This biomarker can aid in early risk stratification and potentially guide preventive measures in high-risk patients. The study emphasizes the clinical utility of arterial lactate as a simple and accessible biomarker for predicting CIN in PCI patients. This approach could enhance early intervention strategies and improve patient outcomes by identifying high-risk individuals.

CONTRIBUTION OF AUTHORS

Research concept- Vishvajit Wakade, Amitesh Nagarwal
Research design- Vishvajit Wakade, Amitesh Nagarwal, Vinod Kr. Poonia
Supervision- Vinod Kr. Poonia, Vishvajit Wakade
Materials- Vishvajit Wakade, Amitesh Nagarwal, Vinod Kr. Poonia
Data collection- Sheshkaran Singh Charan, Manna Bhattacharjee
Data analysis and interpretation- Amitesh Nagarwal, Vishvajit Wakade
Literature search- Vishvajit Wakade, Manna Bhattacharjee
Writing article- Vishvajit Wakade, Manna Bhattacharjee
Critical review- Vishvajit Wakade
Article editing- Sheshkaran Singh Charan, Vishal Gaurab
Final approval- Amitesh Nagarwal

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