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# THE RELATIONSHIP BETWEEN SERUM LACTATE LEVELS AND THE OCCURRENCE OF CONTRAST-INDUCED ACUTE KIDNEY INJURY AND PATIENTS' LONG-TERM PROGNOSIS, IN THE MIDST OF AN EMERGENCY CORONARY ARTERY PERCUTANEOUS INTERVENTION

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**ABSTRACT**

The third leading cause of hospital-acquired acute renal failure is iatrogenic renal impairment caused by contrast media. Contrast-induced acute kidney injury (CI-AKI) is linked to hospital mortality, as well as one- and two-year mortality. An increase in lactate indicators over time indicates negative clinical outcomes. Lactate is a good microcirculation biomarker because it is closely related to capillary perfusion. This was a prospective and registration research project. From March 2016 to March 2017, 250 AMI participants who underwent emergency PCI at Hospital were chosen. The occurrence of CI-AKI was determined by univariate analysis. The accuracy and best cut-off values of lactate for predicting CI-AKI were determined using the receiver operating characteristic (ROC) curve and the corresponding area under curve (AUC). The survival analysis was performed using the Kaplan–Meier method, and the log rank test was used to compare the two groups. Using multivariate Cox regression analysis combined with the backward stepwise method, the predictors of clinical outcomes in AMI patients during a 1-year follow-up after PCI were investigated. Preoperative high lactate levels may be an independent risk factor in patients who develop CI-AKI and an important predictor of long-term poor cardiorenal outcomes with AMI undergoing emergency PCI, according to the findings of this study. More large samples and prospective randomized controlled trials in other institutional settings are needed to confirm this finding.

**Key words** Coronary Artery Percutaneous Intervention, Acute Kidney Injury, Long-Term Prognosis.

## INTRODUCTION

The third leading cause of hospital-acquired acute renal failure is iatrogenic renal impairment caused by contrast media [1]. Contrast-induced acute kidney injury (CI-AKI) is linked to hospital mortality, as well as one- and two-year mortality [2, 3]. An increase in lactate indicators over time indicates negative clinical outcomes [4]. Lactate is a good microcirculation biomarker because it is closely related to capillary perfusion [5, 6].

Blood lactate can reflect tissue oxygen supply and metabolism, as well as a lack of perfusion, which could be linked to the development of CIAKI [7, 8]. Acute changes in hemodynamics may cause changes in blood lactate due to impaired cardiac function in acute myocardial infarction (AMI) [9, 10]. The goal of this study was to see how lactate levels affected the occurrence of CI-AKI and long-term prognosis in AMI patients who underwent emergency percutaneous coronary intervention (PCI) [11, 12].

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**Aim and Objective:**

Lactate and contrast-induced acute kidney injury (CI-AKI) have not been proven to be linked. The goal of this prospective study was to see how lactate levels affected the occurrence of CI-AKI and long-term prognosis in patients with acute myocardial infarction (AMI) who underwent emergency percutaneous coronary intervention (PCI).

**Material and Methods:**

This was a prospective and registration research project. From March 2016 to March 2017, 250 AMI participants who underwent emergency PCI at Hospital Affiliated were chosen. The following were the admission criteria: (1) a diagnosis of ST-segment elevation myocardial infarction (STEMI) that followed the Cardiovascular Diseases Branch of the Chinese Medical Association's 2015 guidelines for the diagnosis and treatment of acute ST-segment elevation myocardial infarction; (2) a diagnosis of non-acute ST-segment elevation myocardial infarction (NSTEMI) that followed the 2015 guidelines for the diagnosis and treatment of acute ST-se (1) hemodynamic instability prior to emergency PCI; (2) long-term renal replacement therapy (including hemodialysis and peritoneal dialysis); renal transplantation; (3) asthma attack, chronic obstructive pulmonary disease, pulmonary fibrosis, and pulmonary heart disease; (4) diabetic ketoacidosis; (5) malignant tumors; (6) CT, MRI, and other contrast agent examinations In the following STEMI and NSTEMI patients, emergency PCI is performed. (1) STEMI patients with chest pain within 12 hours of onset or new left bundle branch block; and (2) patients with clinical and/or electrocardiographic evidence of progressive ischemia within 12 to 24 hours of onset should be considered for emergency PCI. NSTEMI patients with a high risk of myocardial ischemia, as well as those with a GRACE score of >140 and a slew of other high-risk factors. The statistical analysis was performed using the SPSS 19.0 statistical software. The comparison between groups was expressed using an independent sample t-test or a Mann–Whitney U–test, as appropriate, and the measurement data were expressed as mean + standard deviation or median and interquartile range. The percentages were used to express the counting data, and the comparison between groups was done using chi-square analysis or Fisher's exact factors. The risk factors that might affect were then investigated using a multivariable logistic regression model. After univariate analysis, the occurrence of CI-AKI the receiver operating characteristic (ROC) curve and the corresponding area under curve (AUC) were used to determine the accuracy and best cut-off values of lactate for predicting CI-AKI. The Kaplan–Meier method was used for the survival analysis, and the log rank test was used to compare the two groups. The predictive factors of clinical outcomes in AMI patients during 1-year follow-up after PCI were investigated using multivariate Cox regression analysis combined with the backward stepwise method. Statistical significance was defined as a two-sided p-value of less than 0.05.

The occurrence of CI-AKI was determined by univariate analysis. The accuracy and best cut-off values of lactate for predicting CI-AKI were determined using the receiver operating characteristic (ROC) curve and the corresponding area under curve (AUC). The survival analysis was performed using the Kaplan–Meier method, and the log rank test was used to compare the two groups. Using multivariate Cox regression analysis combined with the backward stepwise method, the predictors of clinical outcomes in AMI patients during a 1-year follow-up after PCI were investigated. A two-sided p-value of less than 0.05 was considered statistically significant. There was a significant positive correlation between preoperative lactate and the occurrence of CI-AKI (r=0.416, p0.001), according to Spearman correlation analysis. Age, BMI, and smoking, as well as pre-PCI levels of LVEF and lactate, hemoglobin, albumin, the culprit lesion for LAD, and the use of IABP, were all found to be significantly associated with the development of CI-AKI in univariate regression analysis. After univariate analysis, a multivariable logistic regression model was used. Baseline lactate level and IABP were found to be independent risk factors for the development of CI-AKI in multivariate logistic regression analysis.

This research also has a number of flaws. (1) The conclusion was limited by the fact that it was a single-center, observational study with a small sample size; It was difficult because of the multivariable regression analysis to ensure that differences in baseline characteristics are fully controlled between various groups (2) This study focused solely on a single lactate, and did not track the lactate dynamically. After PCI, there were changes in lactate and creatinine levels (3) Lactate levels are influenced by a variety of factors. Hypotension, stress, and dysregulation of the glycol-metabolic system. Lactate levels may also rise as a result of this. There is only one Lactate measurement can provide information about a patient's hemodynamic condition, but the fact that Lactate levels are lower in patients with high patency rates. It's possible that lactate levels will rise after a successful primary PCI. The levels return to normal. (4) Assessment of renal function. PCI was limited to a change in creatinine level afterward within 72 hours, and long-term renal monitoring. There was no function for a month or even a year. (5) No comparisons were made with other markers in this study. Cystin C, myeloperoxidase, and other proteins, and the mechanism of CI-AKI occurrence was not well understood. (6) We took serum creatinine and lactate levels from patients. During coronary angiography, the baseline Baseline, on the other hand Because an increase in serum creatinine and lactate may occur after admission, these values cannot be considered true baseline values.

| Table 1: Baseline Clinical Characteristics between patients with CI- AKI and those without CI-AKI | | | | |
|---|---|---|---|---|
| Variables | All patients (n=250) | CI- AKI (n=60) | NON- AKI (n=190) | P value |
| Age (years) | 62 ± 12 | 62± 10 | 65 ± 12 | 0.014 |
| Male, n= (%) | 200 (80%) | 50 (20%) | 176 (70.4%) | 0.230 |
| BMI (kg/m2) | 24.5 ± 2.9 | 26.0±3.0 | 25.0 ± 3.2 | 0.008 |
| Systolic BP (mmHg) | 120 ±16 | 130 ±25.0 | 120 ± 24.2 | 0.136 |
| Diastolic BP (mmHg) | 80 ± 16 | 90 ± 20 | 100 ± 25 | 90 ± 35 |
| HR (times /Min) | 75 ± 17 | 80 ± 19 | 82± 20 | 0.077 |
| Smoking, n (%) | 120 (48%) | 60 ± 15.0 | 40 ± 16.0 | 0.289 |
| Hypertension, n (%) | 180(39.2%) | 126 ((50.4%) | 54(21.6%) | 0.657 |
| Diabetes Mellitus, n (%) | 40 ( 16%) | 20 (8%) | 20 (8%) | 0.537 |
| Pre- PCI, n (%) | 25 (10%) | 5 (2%) | 15 (6%) | 0.029 |
| Stroke, n (%) | 15 (6%) | 9 (3.6%) | 6 (2.4%) | 0.182 |
| STEMI, n (%) | 136 (54.4%) | 129 (51.6%) | 7 (2.8%) | 0.476 |
| Lactate (mmol/L) | 2.9 (1.16%) | 3.0 (2.0, 4.6) | 2.6 (2.0, 2.5) | <0.001 |
| WBC (x $10^{9/L}$ | 11.5 (8.9, 11.9) | 9.0 (7.5, 12.6) | 10.8 (8.0, 12.6) | 0.114 |
| Hemoglobin (g/L) | 150 ±20 | 130 ± 25 | 140 ± 20 | 0.024 |
| Albumin (g/L) | 37.0 ± 4.9 | 35.9 ±5.5 | 40 ±4.9 | < 0.001 |
| AST (U/L) | 40 (29.66) | 46 (28.6) | 44 (25.6) | 0.290 |
| Pre-creatinine (umbel/L) | 86.0 ± 30.0 | 95.6± 31.5 | 90.0 ± 26.3 | 0.013 |
| eGFR | 69.0 ± 16.0 | 52.0 ± 12.0 | 17 ± 15.0 | <0.001 |
| **Medication** | | | | |
| IIb/IIIa inhibitors | 225 (90%) | 159 (63.6%) | 66 (26.4%) | 0.155 |
| Beta blockers | 200 (80%) | 150 (60%) | 50 (20%) | 0.201 |
| ACEI/ARBs | 165 (66%) | 105 (42%) | 60 (24%) | 0.772 |
| CCB | 160 (64%) | 115 (46%) | 45 (18%) | 0.676 |
| Statins | 153 (61.2%) | 103 (41.2%) | 50 (20%) | 0.132 |
| Diuretics | 100 (40%) | 60 (24%) | 40 (16%) | 0.339 |
| Contrast dose/ mL | 152 (60.8%) | 116 (46.4%) | 36 (14.4%) | 0.271 |

**Conclusion:**

Preoperative high lactate levels may be an independent risk factor in patients who develop CI-AKI and an important predictor of long-term poor cardiorenal outcomes with AMI undergoing emergency PCI, according to the findings of this study. More large samples and prospective randomized controlled trials in other institutional settings are needed to confirm this finding.

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