Effect of Contrast Media Nephropathy on Renal Function Prognosis in Patients with Acute Myocardial Infarction after PCI

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Research Article

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Abstract

**Purpose** To explore the effect of contrast-induced nephropathy (CIN) on the prognosis of renal function in patients with acute myocardial infarction (AMI) after percutaneous coronary intervention (PCI).

**Methods** This study was a retrospective analysis conducted on 235 patients with acute myocardial infarction who underwent emergency or elective PCI in the Department of Cardiology of the Affiliated Hospital of Jiangsu University from January 2018 to March 2022. We collected the clinical data and laboratory examination data of patients and then divided them into the CIN group and non-CIN group (according to whether CIN had occurred) in order to explore the impact of CIN occurrence on the prognosis of renal function.

**Results** Of the 235 included patients, 28 were in the CIN group and 207 were in the non-CIN group. There were no obvious abnormalities in the clinical data and laboratory examination data of the patients in the two groups before treatment. The glomerular filtration rates of the CIN group vs. the non-CIN group, respectively, were 87.8 ml/min vs. 87.7 ml/min (at admission), 63.4 ml/min vs. 86.6 ml/min (within 3 days of treatment), 64.1 ml/min vs. 83.4 ml/min (within 3 days to 1 month of treatment), and 63.7 ml/min vs. 82.8 ml/min (within 1 month to 1 year of treatment). The incidence of acute renal failure (AKI) in the CIN group was 46.4% higher than that in the non-CIN group (0.5%).

**Conclusion** The short-term and long-term prognosis of AMI patients with CIN is significantly worse than that of patients without CIN. The occurrence of AKI in AMI patients is related to CIN.

Introduction

CIN is a common complication that occurs after coronary intervention in patients with acute myocardial infarction. With an incidence rate of approximately 10%\(^1,2\), it is the third major cause of acute kidney injury (AKI) in hospitals. At present, many studies on CIN have been carried out, including those that have focused on the risk factors of CIN\(^3\) alongside others that have focused on its treatment and prevention\(^4\). Studies have shown that CIN greatly impacts the prognosis of patients, being a predictor of hospital death and late death\(^5,6\). However, there exist different opinions on the short-term and long-term renal function recovery of patients with CIN. Some believe that CIN is transient, such that patients can recover by themselves in a short time frame\(^7\). In contrast, others believe that once kidney injury occurs after PCI, more than half of patients will have sustained kidney injury\(^8\), which is closely related to their mortality at later stages\(^9\). This disagreement may affect the late-stage treatment of patients, thus causing great harm to their life and health. The purpose of this study is therefore to explore the short-term and long-term recovery of renal function in patients with AMI after CIN.

Materials And Methods

**General data.**
We selected 235 patients with acute myocardial infarction who underwent PCI in the Affiliated Hospital of Jiangsu University from January 2018 to March 2022. The inclusion criteria were: (1) patients with acute myocardial infarction who met the diagnostic criteria; (2) patients with existing renal function data taken before PCI, within 3 days after PCI, within 3 days to 1 month after PCI, and within 1 month to 1 year after PCI. The exclusion criteria were: (1) patients with allergies to iodine or iodine contrast agent; (2) patients with phase 5 chronic renal insufficiency or maintenance hemodialysis/peritoneal dialysis; (3) patients with a previous kidney transplant. The study protocol fulfilled the requirements of the Declaration of Helsinki. This study was approved by the Ethics Committee of the Affiliated Hospital of Jiangsu University. Informed consent was obtained from all subjects and/or their legal guardian(s).

**Therapeutic method.**

All patients were loaded with dual antiplatelet therapy before the operation. According to the results of coronary angiography, PCI was only performed on Culprit vessels. Whether to use ACEI/ARB β receptor blockers or calcium antagonists was determined by the attending physician according to each patient's condition. After PCI, 500 ml of 0.9% normal saline was injected intravenously and preventive hydration treatment was performed at a rate of 1 ml/(kg * h). We avoided using nephrotoxic drugs such as quinolones, sulfonamide antibiotics, non-steroidal antipyretics, and chemotherapeutic drugs.

**Data collection.**

We collected the clinical data (age, sex, BMI, systolic blood pressure, diastolic blood pressure, heart rate, type of myocardial infarction, and preoperative hypotension), past medical history (hypertension, diabetes, anemia, smoking, drinking, hyperlipidemia, old myocardial infarction, and PCI), laboratory examination data (albumin, blood urea nitrogen, uric acid, triacylglycerol, cholesterol, low-density lipoprotein, high-density lipoprotein, white blood cell, hemoglobin, hs-CRP, glycosylated hemoglobin, urine routine PH, and LVEF (%)), perioperative medication data (ACEI/ARB β-receptor antagonists, CCB), coronary artery occlusion data (left main artery, left anterior descending branch, left circumflex branch, and right coronary artery), the occurrence of acute renal failure, and both the glomerular filtration rate and blood creatinine levels (at admission, within 3 days of treatment, within 3 days to 1 month of treatment, and within 1 month to 1 year of treatment) of patients.

**Grouping method.**

Patients were divided into the CIN group and non-CIN group (according to whether CIN had occurred) in order to explore the impact of CIN occurrence on the prognosis of renal function.

**Definition of contrast-induced nephropathy.**

Within 72 hours after the contrast agent examination, if the serum creatinine value rose either > 44.2 µmol/L or 25% higher than before the examination, it was determined as CIN\textsuperscript{10,11}.

**Statistical methods.**
For the comparison of data between the two groups, the Student’s t test was used for measurement data, while either the chi-square or the Fisher exact test was used for exact test counting data. The serum creatinine trends of patients at different time periods were represented by a line chart. The relationship between the AKI and CIN of patients was represented by a histogram. All statistical analyses were performed using SPSS statistical software.

Results

Comparison of baseline data between the two groups of patients.

We selected 235 patients with acute myocardial infarction who underwent PCI, including 28 patients with CIN and 207 patients without CIN. From the clinical and laboratory data, there was a statistical difference found in right coronary artery disease between the two groups ($P<0.05$), while there was no significant difference found in the other data (Tables 1 and 2).
Table 1
Comparison of the baseline data between the CIN group and non-CIN group \([x \pm s, n(\%)]\)

<table>
<thead>
<tr>
<th>Baseline data</th>
<th>CIN group</th>
<th>Non-CIN group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>28</td>
<td>207</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>68.0±12.0</td>
<td>64.5±12.5</td>
<td>0.166</td>
</tr>
<tr>
<td>Gender (male), n (%)</td>
<td>18(64.3%)</td>
<td>163(78.7%)</td>
<td>0.088</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>24.7±3.6</td>
<td>25.7±13.8</td>
<td>0.718</td>
</tr>
<tr>
<td>Serum creatinine at admission (µmol/L)</td>
<td>73.4±25.4</td>
<td>78.6±27.9</td>
<td>0.354</td>
</tr>
<tr>
<td>Glomerular filtration rate at admission (ml/min)</td>
<td>87.8±43.5</td>
<td>87.7±35.2</td>
<td>0.985</td>
</tr>
<tr>
<td>Systolic pressure (mmHg)</td>
<td>125.1±19.5</td>
<td>126.3±21.4</td>
<td>0.766</td>
</tr>
<tr>
<td>Diastolic pressure (mmHg)</td>
<td>74.0±11.5</td>
<td>80.2±49.4</td>
<td>0.510</td>
</tr>
<tr>
<td>Heart rate (times/minute)</td>
<td>81.4±18.0</td>
<td>82.9±16.0</td>
<td>0.644</td>
</tr>
<tr>
<td>Type of myocardial infarction (acute ST-segment elevation myocardial infarction), n (%)</td>
<td>22(78.6%)</td>
<td>140(67.6%)</td>
<td>0.240</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>2(71.4%)</td>
<td>131(63.3%)</td>
<td>0.399</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>10(35.7%)</td>
<td>61(29.5%)</td>
<td>0.499</td>
</tr>
<tr>
<td>Anemia, n (%)</td>
<td>5(17.9%)</td>
<td>22(15.5%)</td>
<td>0.744</td>
</tr>
<tr>
<td>History of smoking, n (%)</td>
<td>12(42.9%)</td>
<td>85(41.1%)</td>
<td>0.856</td>
</tr>
<tr>
<td>History of drinking, n (%)</td>
<td>5(17.9%)</td>
<td>44(21.3%)</td>
<td>0.678</td>
</tr>
<tr>
<td>Hyperlipidemia, n (%)</td>
<td>17(60.7%)</td>
<td>106(51.7%)</td>
<td>0.371</td>
</tr>
<tr>
<td>Preoperative hypotension, n (%)</td>
<td>1(3.6%)</td>
<td>7(3.4%)</td>
<td>0.959</td>
</tr>
<tr>
<td>Old myocardial infarction, n (%)</td>
<td>1(3.6%)</td>
<td>3(1.4%)</td>
<td>0.415</td>
</tr>
<tr>
<td>History of PCI, n (%)</td>
<td>2(7.1%)</td>
<td>15(7.2%)</td>
<td>0.984</td>
</tr>
<tr>
<td>ACEI/ARB, n (%)</td>
<td>23(82.1%)</td>
<td>175(84.5%)</td>
<td>0.744</td>
</tr>
<tr>
<td>β-receptor antagonist, n (%)</td>
<td>26(92.9%)</td>
<td>189(91.3%)</td>
<td>0.782</td>
</tr>
<tr>
<td>CCB, n (%)</td>
<td>4(14.3%)</td>
<td>34(16.4%)</td>
<td>0.773</td>
</tr>
</tbody>
</table>
Table 2
Results of the laboratory data and coronary angiography results in the CIN group and non-CIN group [x ± s, n (%)]

<table>
<thead>
<tr>
<th>Baseline data</th>
<th>CIN group</th>
<th>Non-CIN group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>28</td>
<td>207</td>
<td></td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>37.4±3.1</td>
<td>37.0±4.7</td>
<td>0.674</td>
</tr>
<tr>
<td>Blood urea nitrogen (mmol/L)</td>
<td>6.5±2.7</td>
<td>7.5±11.2</td>
<td>0.636</td>
</tr>
<tr>
<td>Uric acid (mmol/L)</td>
<td>318.8±108.2</td>
<td>333.0±112.7</td>
<td>0.530</td>
</tr>
<tr>
<td>Triacylglycerol (mmol/L)</td>
<td>1.7±1.0</td>
<td>3.5±15.6</td>
<td>0.542</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.8±1.0</td>
<td>4.7±1.1</td>
<td>0.731</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>1.2±0.5</td>
<td>1.1±0.3</td>
<td>0.153</td>
</tr>
<tr>
<td>LDL-C (mmol/L)</td>
<td>2.9±0.8</td>
<td>2.8±0.9</td>
<td>0.770</td>
</tr>
<tr>
<td>White blood cell (10^9)</td>
<td>9.4±3.3</td>
<td>10.2±6.4</td>
<td>0.470</td>
</tr>
<tr>
<td>Hemoglobin (g/L)</td>
<td>129.1±16.1</td>
<td>135.1±18.6</td>
<td>0.106</td>
</tr>
<tr>
<td>Hs-CRP (mmol/L)</td>
<td>15.2±30.2</td>
<td>11.8±27.1</td>
<td>0.540</td>
</tr>
<tr>
<td>Glycosylated hemoglobin (%)</td>
<td>6.7±1.6</td>
<td>6.6±1.5</td>
<td>0.587</td>
</tr>
<tr>
<td>Urine routine PH</td>
<td>6.1±0.6</td>
<td>6.2±0.5</td>
<td>0.799</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>58.2±10.8</td>
<td>60.6±10.1</td>
<td>0.248</td>
</tr>
<tr>
<td>Left trunk, n (%)</td>
<td>5(17.9%)</td>
<td>16(7.7%)</td>
<td>0.078</td>
</tr>
<tr>
<td>Left front descending branch, n (%)</td>
<td>27(96.4%)</td>
<td>183(88.4%)</td>
<td>0.196</td>
</tr>
<tr>
<td>Left circumflex branch, n (%)</td>
<td>20(71.4%)</td>
<td>154(74.4%)</td>
<td>0.737</td>
</tr>
<tr>
<td>Right coronary artery, n (%)</td>
<td>18(64.3%)</td>
<td>169(81.6%)</td>
<td>0.033</td>
</tr>
</tbody>
</table>
The glomerular filtration rate of the CIN group was 87.8 ml/min (at admission), 63.4 ml/min (within 3 days of treatment), 64.1 ml/min (within 3 days to 1 month of treatment), and 63.7 ml/min (within 1 month to 1 year of treatment). The corresponding serum creatinine values were 73.4 µmol/L (at admission), 109.3 µmol/L (within 3 days of treatment), 110.5 µmol/L (within 3 days to 1 month of treatment), and 103.0 µmol/L (within 1 month to 1 year of treatment). In the non-CIN group, the glomerular filtration rate was 87.7 ml/min (at admission), 86.6 ml/min (within 3 days of treatment), 83.4 ml/min (within 3 days to 1 month of treatment), and 82.8 ml/min (within 1 month to 1 year of treatment). The corresponding serum creatinine values were 78.6 µmol/L (at admission), 78.8 µmol/L (within 3 days of treatment), 82.3 µmol/L (within 3 days to 1 month of treatment), and 82.1 µmol/L (within 1 month to 1 year of treatment). From the whole line chart, the renal function of the patients in the two groups was not significantly abnormal at the time of admission. However, as time went on, the renal function of the patients with CIN was significantly worse than that of the patients without CIN in both the short-term and long-term, and the difference was statistically significant ($P<0.05$) (Table 3 and Fig. 1).

### Table 3
Renal function in the CIN group and non-CIN group $[x \pm s, n(\%)]$

<table>
<thead>
<tr>
<th>Baseline data</th>
<th>CIN group</th>
<th>Non-CIN group</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>28</td>
<td>207</td>
<td></td>
</tr>
<tr>
<td>Serum creatinine at admission (ummol/L)</td>
<td>73.4±25.4</td>
<td>78.6±27.9</td>
<td>0.354</td>
</tr>
<tr>
<td>Serum creatinine within 3 days of treatment (ummol/L)</td>
<td>109.3±53.5</td>
<td>78.8±21.4</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Serum creatinine within 3 days to 1 month of treatment (ummol/L)</td>
<td>110.5±48.6</td>
<td>82.3±25.6</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Serum creatinine from 1 month to 1 year after treatment (ummol/L)</td>
<td>103.0±40.3</td>
<td>82.1±25.0</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Glomerular filtration rate at admission (ml/min)</td>
<td>87.8±43.5</td>
<td>87.7±35.2</td>
<td>0.985</td>
</tr>
<tr>
<td>Glomerular filtration rate within 3 days of treatment (ml/min)</td>
<td>63.4±34.4</td>
<td>86.6±34.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Glomerular filtration rate within 3 days to 1 month of treatment (ml/min)</td>
<td>64.1±39.6</td>
<td>83.4±32.6</td>
<td>0.005</td>
</tr>
<tr>
<td>Glomerular filtration rate from 1 month to 1 year after treatment (ml/min)</td>
<td>63.7±33.1</td>
<td>82.8±31.6</td>
<td>0.003</td>
</tr>
</tbody>
</table>

AKI occurred in 13 of the 28 patients with CIN, accounting for 46.4%; AKI occurred in 1 of the 207 non-CIN patients, accounting for 0.5%. The difference was statistically significant ($P<0.05$) (Table 4 and Fig. 2).
Table 4
AKI incidence in the CIN group and non-CIN group [x ±s, n (%)]

<table>
<thead>
<tr>
<th>Baseline data</th>
<th>CIN group</th>
<th>Non-CIN group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>28</td>
<td>207</td>
<td></td>
</tr>
<tr>
<td>Acute renal failure, n (%)</td>
<td>13(46.4%)</td>
<td>1(0.5%)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Discussion

The purpose of this study was to explore the effect of CIN on short-term and long-term renal function in patients with acute myocardial infarction after PCI. In this study, the incidence rate of CIN in patients was 11.9%. Many studies have shown that the long-term mortality and prognosis of patients with AMI are both closely related to CIN\(^{12,13}\). Moreover, the more serious that CIN is, the worse the prognosis, the worse the renal function\(^{14}\), and the higher the mortality rate\(^{15}\). In turn, the worse the early renal function is, the lower the success rate of PCI is, and hence, the worse the prognosis is\(^{16,17}\). The study shows that the mortality of AMI patients after PCI is closely related to their later renal function. The worse the recovery of their later renal function, the higher the risk of mortality\(^9\). Even if the hemodynamics and left ventricular ejection fraction are normal after PCI, the mortality of CIN patients is still higher than that of non-CIN patients\(^{18}\). However, some researchers believe that even if CIN occurs in patients with AMI after PCI, it is unrelated to the deterioration of renal function in patients at the later stage, even if previous renal insufficiency does not increase the risk of CIN\(^{19}\). We collected eligible samples over the past four years. No significant difference was found in preoperative renal function, the clinical data, or the laboratory test results between patients in the CIN group and those in the non-CIN group. However, the patients in the CIN group had significantly worse outcomes than those in the non-CIN group in terms of both short-term and long-term renal function, indicating that CIN is a predictor of renal function in patients with AMI after PCI. Once CIN occurred, the renal function of patients in the later stage was significantly worse than that of patients without CIN. The reason for this may be that the contrast agent itself is cytotoxic, thereby exerting a destructive effect on renal tubular epithelial cells and vascular endothelial cells, causing contraction of the entering arterioles, reducing the glomerular filtration rate, and leading to renal ischemia and hypoxia, especially severe renal medulla\(^{20}\). This kind of renal ischemia leads to the further contraction of the afferent arterioles, thereby initiating a vicious cycle\(^{21}\). Once CIN occurs, it indicates that the kidney is seriously damaged, and the recovery of such damage is often limited, which is likely to lead to continuous damage to renal function.

This study showed that the incidence of AKI in CIN patients was 46.4%, while the incidence of AKI in the non-CIN group was 0.5%. The finding reveals a close relationship between CIN and AKI. The two conditions are not only different in the ways that they involve a reduction in renal function; once patients have CIN, its occurrence is highly likely to predict the subsequent occurrence of AKI. CIN means that the serum creatinine increased by more than 25% or more than 44.2umol/L within 3 days, while AKI means that the serum creatinine increased by more than 50% within 1 week. Once the patient has CIN, it
indicates that the patient's renal function is compromised. The cytotoxicity of the contrast agent combined with changes in renal hemodynamics may lead to the further deterioration of the patient's renal function, which is highly likely to lead to AKI. At present, some studies have shown that conducting hemodialysis immediately after the use of contrast media is effective, especially for patients with chronic renal insufficiency. Previous research has shown that the prognosis of renal function in a dialysis group was found to be significantly improved compared with that in a non-dialysis group\textsuperscript{22,23}. However, some object to the validity of these findings, stating that even preventive dialysis will fail to have any impact on the later renal function of patients\textsuperscript{24}.

Some studies have shown that even the small change in serum creatinine that occurs from 12 hours to 24 hours after PCI is a useful indicator to predict later renal function\textsuperscript{8}. The current study is concerned with the impact of CIN on the prognosis of renal function. Compared to the change in renal function that occurs from 12 hours to 24 hours after operation, cardiac physicians pay relatively more attention to the occurrence of CIN within the same timeframe. The diagnosis of this disease is relatively simple and is highly important for the prediction of later renal function. From our research results, we found that the short-term serum creatinine value of patients with CIN dropped temporarily, but these patients do not usually stay in the hospital for a long time. It is often left to the outpatient department to review patients’ renal function. The renal function of patients with CIN tends not to improve significantly, being significantly worse than that of patients without CIN. Therefore, it is advised that patients with CIN are treated differently from those without CIN, especially for renal function. The recovery of renal function is closely related to the overall prognosis and mortality of patients with AMI\textsuperscript{9}. This knowledge is beneficial for identifying patients with poor renal function prognosis in the early stage of exposure to contrast media, thereby allowing treating physicians to conduct timely and effective targeted treatments to improve their prognosis.

This study had some limitations. First, this was a retrospective medical study, in which the number of CIN patients was relatively small, and all patients were from a medical center. Secondly, we recommend that a future study be conducted to collect renal function data for a longer time, such as up until one year after PCI, so that the results will be more reliable.

**Conclusions**

The prognosis of renal function in patients with CIN after PCI in AMI patients was significantly poor. AMI patients with CIN are more prone to AKI. Therefore, it is particularly important to monitor the renal function of AMI patients with CIN after the operation.

**Abbreviations**

CIN: contrast-induced nephropathy; AMI: acute myocardial infarction; PCI: percutaneous coronary intervention; AKI: acute kidney injury.
Declarations

Acknowledgements

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Author contributions

All authors meet the following criteria: 1. Design papers and analysis data. 2. Draft articles or make critical amendments to them. 3. All authors have reviewed the manuscript.

Funding

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Availability of data and materials

All data generated or analysed during this study are included in this published article.

Ethics approval and consent to participate

The study protocol fulfilled the requirements of the Declaration of Helsinki. This study was approved by the Ethics Committee of the Affiliated Hospital of Jiangsu University. Written informed consent was obtained from all patients included in the study. (Ethics Approval no. KY2023K0303)

Consent for Publication

Not applicable.

Competing interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Author details

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References


**Figures**
Figure 1

Time1 indicates the glomerular filtration rate at admission; time2 indicates the glomerular filtration rate within 3 days of treatment; time3 indicates the glomerular filtration rate within 3 days to 1 month of treatment; time4 indicates the glomerular filtration rate from 1 month to 1 year after treatment.
Figure 2

CIN indicates contrast-induced nephropathy. AKI indicates acute kidney injury.