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*Original Article*

# The prophylactic effect of alprostadil on contrast-induced nephropathy in renal insufficiency patients after percutaneous coronary intervention

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**Abstract:** Objectives: This study explored and analyzed the prophylactic effect of Alprostadil on contrast-induced nephropathy (CIN) after percutaneous coronary intervention (PCI) in patients with renal insufficiency. Methods: From June 2014 to June 2017, 156 patients with coronary artery disease and renal insufficiency who electively underwent PCI in our hospital were enrolled and randomly divided into Alprostadil group (n=77) and placebo control group (n=79) by random number table. The control group was given hydration and placebo, while the observation group received hydration and Alprostadil. The alprostadil and placebo were infused intravenously 30-90 minutes before operation until four hours after surgery. The changes of biochemical indicators, serum creatinine (Scr), glomerular filtration rate (GFR), the level of neutrophil gelatinase-associated lipocalin (NGAL) in urinary neutrophils, and the incidence of CIN were compared between the two groups of patients before and after PCI surgery. Results: The difference of SCr and GFR between the two groups of patients was statistical insignificant before and after PCI surgery (*P>0.05*), while the level of NGAL in both groups 12 h and 24 h after operation were critically higher than those prior-operation (*P<0.05*), and the increase in the control group was more obvious (*P<0.05*). The serum CysC, Hcy, and hs-CRP levels of the two groups 24 h after surgery were remarkably higher than those before surgery (*P<0.05*), and the indicators in observation group were superior to those in control group after surgery (*P<0.05*). The difference between the two groups of patients in incidence of CIN primary endpoint was statistically insignificant (*P>0.05*), while the incidence of secondary endpoint of CIN ≥ 0.3 mg/dL in Alprostadil group was significantly lower than that in control group (*P<0.05*). Conclusion: For renal insufficiency patients undergoing PCI, the associative usage of Alpromazil with routine treatment can effectively prevent CIN and is worthy of clinical promotion.

**Keywords:** Alprostadil, renal insufficiency, percutaneous coronary intervention (PCI), contrast-induced nephropathy (CIN), prevention

## Introduction

The incidence rate of contrast-induced nephropathy (CIN) has been increasing year by year with the rise of patients undergoing coronary angiography, as well as the prolonged operation time and the increased consumption of contrast medium [1, 2]. CIN refers to the increase of serum creatinine over 44.2 μmol/L (0.5 mg/dl) or exceeds 25% of the basic value 48 hours after radiography, and the kidney damage that excluding other causes [3]. In general, the incidence of CIN is about 1.2-3.0%, but it may increase in high-risk patients with CIN risk factors such as advanced age and renal insufficiency. A few patients need to receive dialysis treatment, seriously affecting their prognosis [4]. Therefore, for high-risk patients with renal insufficiency, it is particularly crucial to seek effective measures to prevent CIN. Alprostadil is a kind of natural prostaglandin substance. It can promote the increase of concentration of cyclic adenosine monophosphate in cells by regulating the activities of adenylate cyclase and phosphodiesterase, activates a series of protein kinases that depend on cyclic adenosine monophosphate to dilate blood vessels, and improve the peripheral blood circulation and renal blood flow. Alprostadil can reduce residual glomerular pressure, high filtration and high perfusion, inhibit the release of inflammatory mediators

IL-1, TNF-α, etc., reduce the formation of antigen-antibody reactions, inhibit aggregation of platelets and the formation of atherosclerotic plaque, and reduce urine protein. Therefore, scholars believe that Alprostadil can play a positive role in the prevention of CIN [5, 6]. This study, aiming at CIN high-risk patients with renal insufficiency, analyzed the preventive effect of Alprostadil on CIN after PCI treatment in patients with renal insufficiency.

## Materials and methods

### *Clinical data*

From June 2014 to June 2017, 156 patients with coronary artery disease and renal insufficiency who underwent PCI in our hospital were enrolled and randomly divided into Alprostadil group (n=77) and placebo control group (n=79) by random number table. This study obtained the approval from the ethics committee of our hospital.

### *The inclusive criteria and exclusive criteria*

Inclusive criteria: (1) The patients met the diagnostic criteria for coronary heart disease and had indications for receiving PCI treatment [7]; (2) Patients met the criteria of renal insufficiency: serum creatinine clearance rate ≥ 104 mmol/L in the latest half year; (3) The informed consent had been signed voluntarily by the patients.

Exclusive criteria: (1) Dialysis patients with shock, systolic blood pressure <95 mmHg, acute renal insufficiency or chronic renal insufficiency; (2) Patients who received intravenous contrast media 10 days before enrollment and needed contrast media 6 days after admission; (3) Patients received nephrotoxic medicine during perioperative period; (4) Patients with malignant tumor; (5) Patients with severe heart failure; or (6) Patients with thyroid or adrenal dysfunction.

### *Methods*

All patients were given intravenous saline hydration at least 4 h before the use of contrast agent until 12 h after the operation, and blood samples were taken before vein hydration. The patients were treated with non-ionic contrast medium (iobitridol injection and iohexol, France). The physicians used the postoperative interventional drugs according to patients' conditions, and the specific drug administration method was referred to the Spargias trial [8]. Intravenous hydration of the two groups was performed by 1.5 $ml \cdot kg^{-1} \cdot h^{-1}$ continuous intravenous drip. The observation group, on the basis of hydration, was given Alprostadil (Liaoning Green Biopharmaceutical Group Co., Ltd., H20066828) 1 $ng \cdot kg^{-1} \cdot min^{-1}$ with intravenous pump 30-90 minutes before the use of the contrast agent, and continued until 4 h after the use of the contrast agent. The vital signs were monitored during the application of Alprostadil, and any cases of bleeding or systolic blood pressure <90 mmHg were excluded from the test. The control group was given an equal volume of saline on the basis of hydration, which was pumped slowly at the same speed. Both groups were encouraged to drink water after surgery.

### *Observation of indexes*

Measurement of serum creatinine (SCr) and other biochemical indicators: We collected the peripheral venous blood of patients before surgery and 12 h, 24 h, 48 h postoperatively, and detected the SCr and biochemical indexes by automatic biochemical analyzer, as well as the changes of serum CysC, Hcy, and hs-CRP.

Measurement of neutrophil gelatinase-associated lipocalin (NGAL): Urine samples were collected before surgery and 12 h, 24 h and 48 h after surgery, centrifuged at 3000 r/min for 20 min (centrifugation radius 16 cm), and the supernatant was stored in a refrigerator at -80°C. The measurement was carried out by the same testing personnel with ELISA method, and the procedures were in strict accordance with the kit instructions.

Calculation of glomerular filtration rate (GFR): GFR was calculated by Modification of Diet in Renal Disease (MDRD formula): GFR ($ml \cdot min^{-1} \cdot 1.73m^{-2}$) = $175 \times [SCr\ (mg/dl)]^{-1.234} \times [age\ (years\ old)]^{-0.179} \times 0.79$ (female).

### *Diagnostic criteria of CIN*

The primary endpoint of CIN: The absolute increase in postoperative serum creatinine concentration from the baseline level 1 d and 5 d after surgery was ≥ 0.5 mg/dl, or the relative increase was ≥ 25%.

The secondary endpoint of CIN: The absolute increase in postoperative serum creatinine concentration from the baseline level was ≥ 0.3 mg/dL or ≥ 0.5 mg/dl, or the relative increase was at least ≥ 25%.

*Statistical analysis*

Data processing and analysis were performed by statistical software SPSS 22.0. The measurement data was expressed by mean ± standard deviation ($\bar{x}$ ± sd), and the comparison of measurement data was by *t*-test. The technical data were expressed as percentage, and the count data was compared by $\chi^2$ test. *P<0.05* was considered as statistically significant.

## Results

*Comparison of general condition*

The differences of clinical data, baseline biochemical indicators, past medical history and drug usage between the two groups were statistically insignificant (P>0.05), as shown in **Table 1**.

*Comparison of blood SCr, GFR and NGAL between the two groups before and after PCI*

The difference of SCr and GFR between the two groups of patients was statistical insignificant before and after PCI surgery (*P>0.05*), while the levels of NGAL in both groups 12 h and 24 h after operation were critically higher than those prior-operation (*P<0.05*), and the increase in the control group was more obvious (*P<0.05*), as shown in **Table 2** and **Figure 1**.

*Comparison of changes in serum Cysc, Hcy, hs-CRP between the two groups before and after PCI*

The serum CysC, Hcy, and hs-CRP levels of the two groups 24 h after surgery were remarkably higher than those before surgery (*P<0.05*), and the indicators in observation group were superior to those in control group after surgery (*P<0.05*), as shown in **Table 3**.

*Comparison of CIN incidence between the two groups*

The difference between the two groups of patients in incidence of CIN primary endpoint was statistically insignificant (*P>0.05*), while the incidence of secondary endpoint of CIN ≥ 0.3 mg/dL in Alprostadil group was significantly lower than that in control group (*P<0.05*), as shown in **Table 4**.

## Discussion

CIN is a major complication caused by the application of iodine contrast medium and is also the third most common cause of iatrogenic-acquired renal failure. Therefore, the prevention and treatment of CIN have been given great attention by clinicians [9]. Currently, it is generally believed that the primary mechanism of CIN is the hemodynamic changes caused by contrast medium and the direct toxic effects on renal tubular epithelial cells that produces a large number of free radicals and aggravate the damage of renal function [10, 11]. The imbalance of vasoconstriction caused by the contrast medium can lead to the contraction of the glomerular arterioles, the reduction of renal blood flow, the ischemic injury of tissues or even cell necrosis [12]. A large number of free radicals generated during ischemia-reperfusion can promote the toxicity of renal tubular epithelial cells, reduce the activity of antioxidant enzymes and peroxide-activating enzymes, and remarkably increase lipid peroxidation, which further leads to the renal tubular damage [13-15]. Therefore, as important physicochemical indexes, the osmotic pressure and viscosity of contrast medium have been widely concerned in clinic practice.

The only recognized way at present to effectively prevent CIN is hydration therapy. It can reduce the viscosity of contrast medium and the hyperosmotic state caused by subclinical dehydration. The hydration therapy has the effect of antagonizing the renin-angiotensin system, thereby reducing tube ball feedback, slowing the contraction of renal blood flow, increasing the amount of urine to prevent the obstruction of renal tubules and reducing the shrinkage of vascular material generated. Therefore, renal medullary ischemia can be alleviated and the toxicity of contrast agent to renal tubular epithelial cells can be reduced directly [16-18].

Alprostadil is a natural prostaglandin with strong vasodilator effect. It plays a very important role in the maintenance and distribution of renal blood flow, as well as the excretion of

**Table 1.** Comparison of general condition and operation status of the two groups of patients

| Material | Alprostadil group (n=77) | Control group (n=79) | $t/\chi^2$ | *P* |
|---|---|---|---|---|
| Gender | | | | |
| Male | 41 | 45 | 0.218 | 0.641 |
| Female | 36 | 34 | | |
| Age (years, $\bar{x}$ ± sd) | 68.18±11.25 | 66.45±12.12 | 0.923 | 0.357 |
| Body mass index (kg/m$^2$, $\bar{x}$ ± sd) | 26.33±2.55 | 26.68±2.87 | 0.805 | 0.422 |
| LVEF ($\bar{x}$ ± sd) | 46.90±1.12 | 46.95±1.57 | 0.229 | 0.820 |
| Systolic pressure (mmHg, $\bar{x}$ ± sd) | 137.96±15.60 | 136.97±13.68 | 0.422 | 0.674 |
| Diastolic pressure (mmHg, $\bar{x}$ ± sd) | 78.64±8.27 | 79.82±9.20 | 0.842 | 0.401 |
| Dosage of contrast medium (ml, $\bar{x}$ ± sd) | 258.22±58.72 | 247.42±64.33 | 1.094 | 0.276 |
| Duration of venous hydration (h, $\bar{x}$ ± sd) | 17.16±1.18 | 17.57±1.68 | 1.760 | 0.080 |
| Hydration length before use of contrast medium (h ,$\bar{x}$ ± sd) | 5.63±0.51 | 5.53±0.64 | 1.078 | 0.283 |
| Total venous hydration (ml, $\bar{x}$ ± sd) | 2224.78±163.45 | 2211.62±141.36 | 0.538 | 0.591 |
| Total amount of hydration before use of contrast medium (ml, $\bar{x}$ ± sd) | 640.83±51.26 | 646.22±57.84 | 0.615 | 0.539 |
| Baseline erythrocyte volume (fL, $\bar{x}$ ± sd) | 38.05±5.28 | 38.19±6.04 | 0.154 | 0.878 |
| Mehran nephropathy Risk score (point, $\bar{x}$ ± sd) | 8.99±1.27 | 9.44±1.78 | 1.814 | 0.072 |
| History of CABG | 1 (1.30) | 3 (3.80) | 0.231 | 0.631 |
| History of PCI, n (%) | 23 (29.87) | 18 (22.78) | 1.010 | 0.315 |
| Diabetes, n (%) | 29 (37.66) | 30 (37.97) | 0.001 | 0.968 |
| Hypertension, n (%) | 69 (89.61) | 71 (89.87) | 0.003 | 0.957 |
| Peripheral arterial disease, n (%) | 9 (11.69) | 10 (12.66) | 0.034 | 0.853 |
| Calcium antagonist, n (%) | 25 (32.467) | 24 (30.38) | 0.079 | 0.779 |
| Loop diuretics, n (%) | 30 (38.96) | 32 (40.51) | 0.039 | 0.844 |
| ACEI/ARB, n (%) | 53 (68.83) | 54 (68.35) | 0.004 | 0.949 |
| Statins, n (%) | 62 (80.52) | 59 (74.68) | 0.763 | 0.382 |
| Acetylcysteine, n (%) | 6 (7.79) | 8 (10.3) | 0.260 | 0.610 |
| II/III Inhibitors, n (%) | 4 (5.19) | 7 (8.86) | 0.800 | 0.371 |
| Heart failure, n (%) | 8 (10.39) | 12 (15.19) | 0.804 | 0.370 |

**Table 2.** Changes of blood SCr, GFR and NGAL between the two groups before and after PCI ($\bar{x}$ ± sd)

| Group | Time | SCr (μmol/L) | GFR (ml·min$^{-1}$·1.73m$^{-2}$) | Urine NGAL (ng/ml) |
|---|---|---|---|---|
| Alprostadil group (n=77) | Before operation | 86.74±21.94 | 83.06±24.83 | 7.42±2.16 |
| | 12 h postoperatively | 89.72±20.73 | 81.96±16.87 | 37.95±17.84$^{*,\#}$ |
| | 24 h postoperatively | 90.22±20.05 | 80.73±21.65 | 16.49±5.92$^{*,\#}$ |
| | 48 h postoperatively | 91.75±21.07 | 82.30±23.41 | 7.63±1.33 |
| | F | 1.464 | 0.638 | 18.382 |
| | P | 0.147 | 0.523 | 0.000 |
| Control group (n=79) | Before operation | 87.04±19.89 | 82.07±25.64 | 7.49±2.10 |
| | 12 h postoperatively | 91.37±18.75 | 81.99±19.78 | 48.95±21.47$^{*}$ |
| | 24 h postoperatively | 90.83±21.53 | 83.41±21.37 | 21.98±6.37$^{*}$ |
| | 48 h postoperatively | 92.06±18.95 | 82.93±23.13 | 7.58±1.21 |
| | F | 1.624 | 0.433 | 22.371 |
| | P | 0.106 | 0.665 | 0.000 |

Note: compared with preoperative, $^{*}$P<0.05; compared with control group, $^{\#}$P<0.05.

electrolytes and water [19, 20]. Previous studies have shown that Alprostadil can prevent CIN. In this study, the researchers adopted placebo-controlled method to evaluate the preventive effect of 10·kg$^{-1}$·min$^{-1}$, 20·kg$^{-1}$·min$^{-1}$ and 40 ng·kg$^{-1}$·min$^{-1}$ Alprostadil respectively on CIN in

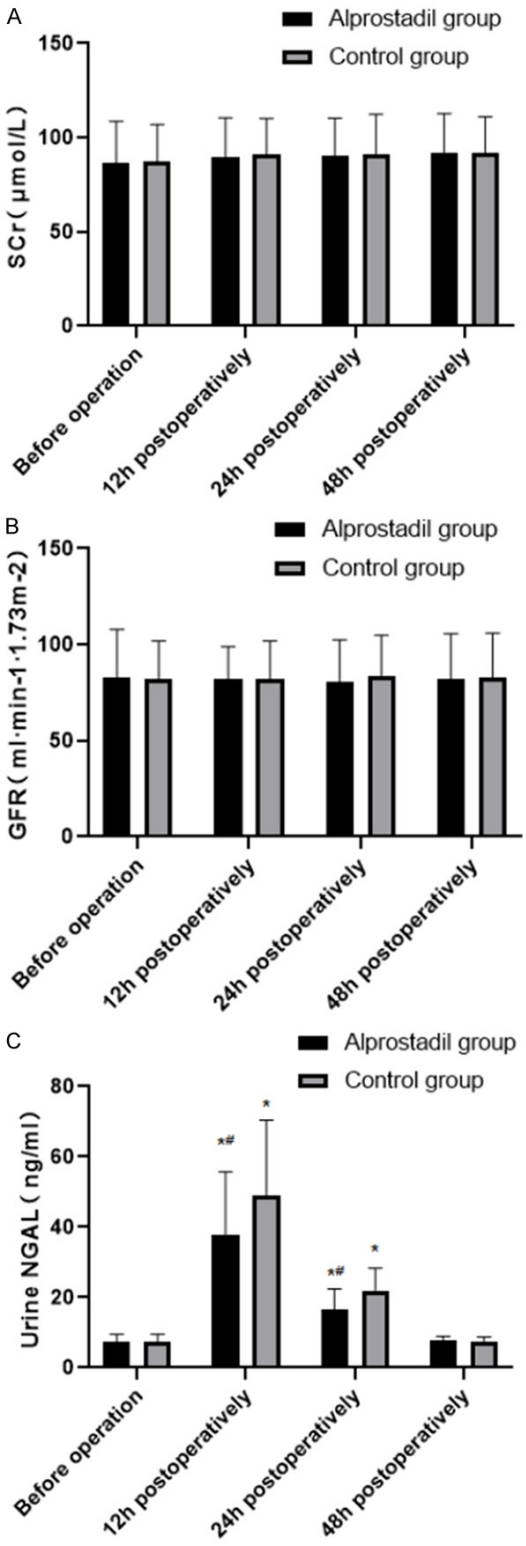


**Figure 1.** Comparison of SCR, GFR and Urine NGAL between the two groups before and after PCI. Note: A: SCR; B: GFR; C: Urine NGAL. Compare with before operation, *P<0.05; Compare with control group, #P<0.05.

patients with renal insufficiency. Compared with the control group, the medium dose of Alprostadil 20 $ng \cdot kg^{-1} \cdot min^{-1}$ can significantly reduce the serum creatinine levels in patients. In the study, three different doses of Alprostadil were evaluated for the preventive effect on CIN. At the same time, Alprostadil was added on the basis of hydration and adopted in safest dosage for the sake of safety [21, 22].

The results of this study indicated that the difference of SCr and GFR between the two groups of patients was statistical insignificant before and after PCI surgery, while the levels of NGAL in both groups 12 h and 24 h after operation were critically higher than those prior-operation, and the increase in the control group was more obvious. Similar to the results of other scholars [23, 24], NGAL is believed to be an early molecular marker reflecting acute kidney injury, which sensitivity is higher than SCr. And the combination of Alprostadil on the basis of hydration therapy has a certain renal protection in the prevention and treatment of CIN. This study observed the primary endpoint of CIN between the two groups of patients. In the control group (n=79 cases), there were 16 cases with contrast nephropathy (20.3%), and in the Alprostadil group (n=77 cases), there were 9 cases with contrast nephropathy (11.7%). There was statistical insignificant difference in the incidence of renal insufficiency in the primary endpoint of CIN between the control group and the alprostadil group in this study. Although there was no statistical difference, the incidence of contrast nephropathy in the control group was higher than that in the Alprostadil group. The absolute value of the serum creatinine concentration of the secondary end point was ≥ 0.3 mg/dL. In control group, there were 20 patients (25.4%) with CIN, and in Alprostadil group the number of CIN cases was 10 (12.3%), P=0.047. The slight increase in serum creatinine concentration in this group of patients indicates that the renal function is less impaired, and demonstrates that the combination of Alprostadil on the basis of hydration can further save the glomerular filtration function, which is similar to the results of other scholars [25, 26].

However, there are shortcoming existed in this study. The sample size of patients included is relatively small, and the mechanism of

**Table 3.** Changes of serum Cysc, Hcy and hs-CRP before and after PCI operation between the two groups ($\bar{x}$ ± sd)

| Group | Time | CysC (mg/L) | Hcy (μmol/L) | hs-CRP (mg/L) |
|---|---|---|---|---|
| Alprostadil group (n=77) | Pre-surgery | 1.84±0.33 | 12.83±3.37 | 4.28±0.79 |
| | 24 h preoperatively | 4.19±1.02*,# | 18.69±4.18*,# | 6.84±1.36*,# |
| | t | 19.235 | 9.577 | 14.283 |
| | P | 0.000 | 0.000 | 0.000 |
| Control group (n=79) | Pre-surgery | 1.81±0.46 | 12.75±2.96 | 4.52±0.82 |
| | 24 h preoperatively | 5.74±1.63* | 23.12±6.44* | 8.22±1.95* |
| | t | 20.624 | 13.004 | 15.546 |
| | P | 0.000 | 0.000 | 0.000 |

Note: compared with pre-surgery, *$^{*}P<0.05$; compared with the control group, $^{\#}P<0.05$.

**Table 4.** Comparison of the incidence of CIN between the two groups

| Group | Case | CIN primary endpoint | CIN ≥ 0.3 mg/dL Secondary endpoint |
|---|---|---|---|
| Alprostadil group | 77 | 9 (11.69) | 10 (12.99) |
| Control group | 79 | 16 (20.25) | 22 (27.85) |
| $\chi^2$ | - | 2.126 | 5.282 |
| P | - | 0.145 | 0.022 |

Alprostadil on CIN prevention and treatment and its further efficacy are still awaiting a multicenter, randomized, double-blind, and large-scale clinical prospective study.

In conclusion, for renal insufficiency patients undergoing PCI, the associative usage of Alpromazil with conventional treatment can effectively prevent CIN caused by contrast agents and is worthy of clinical promotion.

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## Disclosure of conflict of interest

None.

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