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Research Article

Left Ventricular End-Diastolic Pressure and B-Type Natriuretic Peptide Levels Guidance of Low-Dose Furosemide Treatment to Prevent Contrast-Induced Nephropathy in Patients with Percutaneous Coronary Intervention: A Randomized Controlled Trial

Guoqiang Gu, Demin Liu, Rui Lu, and Wei Cui

Department of Cardiology, The Second Hospital of Hebei Medical University, Shijiahzhuang, Hebei 050000, China

Correspondence should be addressed to Wei Cui; cuiweihb2h@163.com

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Objective. We aimed to explore the preventive effect of low-dose furosemide administration guided by left ventricular end-diastolic pressure (LVEDP) and B-type natriuretic peptide (BNP) based on adequate hydration on contrast-induced nephropathy (CIN) in patients with percutaneous coronary intervention (PCI). *Methods*. This parallel randomized clinical trial was conducted at a tertiary hospital in China. A total of 1053 consecutive patients (71.98% men) who underwent PCI at our hospital were enrolled. Pre-PCI plasma BNP levels were recorded. Patients enrolled received a continuous intravenous infusion of normal saline starting 4 h before PCI until 24 h after surgery. LVEDP was measured immediately after surgery. Patients in the control group received intravenous furosemide injection (20 mg). Patients in the experimental group received furosemide if they showed LVEDP ≥15 mmHg, a post-PCI BNP level ≥100 pg/mL, and/or a post-PCI BNP value > 150% of the pre-PCI value. The primary and secondary outcome measures were serum creatinine levels, glomerular filtration rate, and creatinine clearance rate measured before and after PCI. CIN incidence was compared between the two groups. Logistic regression analysis was used to study the risk factors for CIN. *Results*. CIN incidence was significantly higher in the control group than in the experimental group ($P < 0.05$). Logistic regression analysis showed that elevated LVEDP and BNP levels were risk factors. As LVEDP increased, the CIN incidence also increased (odds ratio (OR) 1.038, 95% confidence interval (CI) 1.006–1.070). The OR of BNP was 1.001 (95% CI 1.000–1.002). *Conclusions*. Low-dose furosemide administration guided by LVEDP or BNP is superior to direct low-dose administration on the basis of adequate hydration during PCI. This trial is registered with ChiCTR-IOR-14005250

1. Introduction

Contrast-induced nephropathy (CIN) is a common complication of coronary intervention therapy [1]. CIN is defined as an increase in serum creatinine of more than 25% from the baseline, or a 0.5 mg/dL increase in the absolute serum creatinine value, within 48 hours after administration of contrast agents. In view of its high incidence and long-term adverse effects, researchers have carried out extensive studies on the prevention measures for CIN [2].

Currently, periprocedural hydration is recognized as an effective strategy for preventing CIN [3]. Moreover, our previous studies have demonstrated that low-dose furosemide administered with adequate hydration reduces the occurrence of CIN more than hydration alone [4]. However, it is crucial to assess the sufficiency of hydration, which requires careful monitoring of the effective blood volume in clinical practice.

Left ventricular end-diastolic pressure (LVEDP) is a reference standard indicator for assessing left ventricular preload and can be used to monitor hemodynamic changes.

Studies have shown that the volume of hydration treatment can be monitored by measuring the LVEDP during the periprocedural period and the use of diuretics in patients with increased LVEDP levels can significantly reduce the incidence of CIN [5].

In practice, B-type natriuretic peptide (BNP) testing is important for evaluating heart function and correlates with the changing trend of LVEDP, which can identify heart failure and guide treatment [6]. However, it is unknown whether the combined monitoring of BNP and LVEDP levels can guide the use of diuretics to reduce the incidence of CIN. Furthermore, BNP can indirectly reflect the patient's blood volume status. Although BNP testing is not as accurate as LVEDP, BNP measurements are relatively easy to obtain, which is conducive to a wide range of clinical applications. Therefore, our clinical trial attempted to clarify the significance of LVEDP or BNP levels to serve as a guide for the use of low-dose furosemide in a combined hydration treatment for patients who undergo percutaneous coronary intervention (PCI), with the aim of developing an individualized treatment plan for these patients.

2. Methods

2.1. Study Patients. A total of 1053 consecutive patients with coronary artery disease (CAD) who underwent PCI were enrolled in this clinical trial (Chinese Clinical Trial Registry ChiCTR-IOR-14005250) that was conducted from October 2017 to May 2019. The study was performed at the Second Hospital of Hebei Medical University. The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki, as reflected in a priori approval by the Ethics Committee of Hebei Medical University (approval number: 2018-R289). All patients provided written informed consent.

2.2. Exclusion Criteria. Patients were excluded for the following reasons: (1) allergic to contrast media, (2) exposure to contrast media within the previous 48 h, (3) kidney or heart transplantation, (4) left ventricular thrombus, (5) congestive heart failure (stage IV), and (6) another severe disease that was a contraindication for hydration.

2.3. Study Design and Experimental Procedures. The patients were randomly assigned to either the experimental or control group. The research staff of the study opened the next sequentially numbered opaque envelope that contained the product assignment. An individual unassociated with the clinical portion of the study prepared the envelopes. The randomization sequence was created using Stata 9.0 (Stata Corp., College Station, TX) statistical software and stratified by the center with a 1 : 1 allocation using random block sizes of 2, 4, and 6. The general characteristics of patients and the results of routine examinations were collected. All patients were treated with 0.9% sodium chloride solution at a rate of 1 mL/kg/h at 4 h before surgery until 24 h after surgery. The BNP value of all patients was measured before surgery, and only the experimental group underwent BNP testing after surgery. The LVEDP was directly measured at the end-diastolic phase using a Cordis pigtail catheter placed inside the left ventricle. The control group was administered an intravenous injection of 20 mg furosemide after PCI. However, the administration of furosemide to those in the experimental group was based on the fulfillment of at least one of the following conditions: LVEDP ≥15 mmHg, a post-PCI BNP level ≥100 pg/mL, and a post-PCI BNP value > 150% of the pre-PCI BNP value. The full trial protocol can be accessed in the Chinese Clinical Trial Registry (http://www.chictr.org.cn/abouten.aspx) having registration number ChiCTR-IOR-14005250.

2.4. Evaluation Markers. The serum creatinine (SCr) level was measured before and 48 h after surgery, and the glomerular filtration rate (GFR) and serum creatinine clearance rate (CCr) were calculated as described below. Pre- and postoperative changes in SCr, GFR, and serum CCr values were compared within each group. After the operation, the total fluid intake and urine output at 24 h were carefully recorded. Other blood sample markers, such as routine blood tests and biochemical parameters, and the incidence of CIN, were also compared between these two groups.

2.5. Diagnostic Criteria. CIN was defined as an elevation in SCr of >25% from baseline or >0.5 mg/dL within 48 h of contrast agent administration, with the exclusion of other causes of acute renal injury. The formula for the modification of the diet for renal disease was used to calculate the GFR : $\mathrm{GFR}\ (\mathrm{mL{\bullet}min^{-1}{\bullet}1.73\,m^{-2}}) = 186 \times \mathrm{SCr}\ (\mu\mathrm{mol/L})^{-1.154} \times \mathrm{age}\ (\mathrm{years})^{-0.203} \times (0.742, \text{ if female})$. The Cockcroft-Gault formula was used to calculate the serum CCr : $\mathrm{CCr}\ (\mathrm{mL/min}) = [140 - \mathrm{age}\ (\mathrm{years}) \times \mathrm{weight}\ (\mathrm{kg}) \times (0.85 \text{ if female})]/[72 \times \mathrm{SCr}\ (\mu\mathrm{mol/L})]$.

2.6. Statistical Analyses. SPSS 21.0 software (IBM Corp., Armonk, NY, USA) was used for the statistical analyses. Measurement data are presented as the mean ± standard deviation or median and interquartile range. Classification data are presented as percentages. SCr, serum CCr, and GFR values were compared between the two groups using repeated-measure analysis of covariance. The pre- and post-surgical comparisons within each group were conducted using the rank-sum test. The incidence of CIN in all patients or subgroups was analyzed using the χ^2 test. Logistic regression analysis was used to determine the risk factors for CIN. A P value of <0.05 was considered statistically significant.

2.7. Patient and Public Involvement. It was not appropriate or possible to involve patients or the public in the design, conduct, reporting, or dissemination plans of our research.

3. Results

3.1. General Baseline Characteristics of the Participants. A total of 1053 consecutive patients with CAD who underwent PCI were enrolled. Of the 1053 patients, 758 (71.98%) were men and 295 (28.02%) were women, with an age range of

21–84 years (mean, 59.0 years; SD, 13.0 years), a weight range of 42–135 kg (mean, 70.5 kg; SD, 15.0 kg), and 58 developed CIN. The baseline characteristics of all patients are shown in Table 1.

3.2. Comparison of Baseline Characteristics between the Control and Experimental Groups. There were several significant differences in baseline characteristics between the control and experimental groups, including left ventricular diameter, 24-hour total intake, blood homocysteine level, and C-reactive protein level ($P < 0.05$, Table 1).

No significant differences were observed in other general baseline clinical conditions (age, height, weight, sex, and smoking), concomitant diseases (hypertension, diabetes, chronic renal insufficiency, or hyperlipidemia), laboratory indicators (fasting blood glucose, total cholesterol, albumin, urea nitrogen, serum creatinine, uric acid, cystatin C, $\beta 2$ microglobulin, platelet, and hemoglobin levels), and operative data (number of coronary artery lesions, puncture site, and type/dose of contrast media) ($P > 0.05$, Table 1).

3.3. Diagnostic Markers of the Experimental and Control Groups. In the control group, the postoperative SCr levels were significantly higher than the preoperative levels ($P < 0.05$, Supplementary Table S1), and the GFR and serum CCr values significantly decreased after surgery ($P < 0.05$, Supplementary Table S1).

In the experimental group, the postoperative SCr values decreased slightly from preoperative levels, although the difference was not significant (Supplementary Table S1). The GFR and serum CCr values increased after surgery, although these differences were not significant (Supplementary Table S1).

3.4. Incidence of CIN. The incidence of CIN was significantly higher in the control group than in the experimental group (8.14% and 2.86%, respectively; $P < 0.05$, Table 2).

Patients in the control group received intravenous furosemide injection (20 mg). Patients in the experimental group received furosemide if they showed LVEDP ≥15 mmHg, a post-PCI BNP level ≥100 pg/mL, and/or a post-PCI BNP value of >150% of the pre-PCI value. The incidence of CIN in subgroups of the experimental group was not significant ($P > 0.05$, Table 3).

3.5. Logistic Regression Analysis. Binary logistic regression analysis was performed to further explore the predictive value of LVEDP and BNP for CIN. The results showed that, for LVEDP, the odds ratio (OR) was 1.038, with a 95% confidence interval (CI) of (1.006, 1.070), ($P = 0.020$). For BNP, the OR was 1.001, with a 95% CI of (1.000, 1.002), ($P = 0.021$). These findings indicate that the incidence of CIN increased with an increase in the LVEDP and BNP values (Supplementary Table S2).

The receiver operating characteristic curve analysis of the predictive performance of LVEDP for CIN produced an area under the curve value of 0.606, with a sensitivity of 39.2% and specificity of 77.1% ($P = 0.013$) (Figure 1). The optimal LVEDP cut-off for the prediction of CIN was 15.5 mmHg. The area under the curve value for BNP was 0.605, with an optimal cut-off value of 116.5 pg/mL, a sensitivity of 40.0%, and a specificity of 83.7% ($P = 0.014$) (Figure 1).

4. Discussion

We found that under the same hydration plan, LVEDP and BNP measurements in the experimental group were significantly lower than those with the conventional administration of furosemide in the control group. Logistic regression analysis showed that increased LVEDP and BNP levels were positively correlated with the occurrence of CIN. The following reasons potentially contribute to the findings of this study: (1) all patients in the control group were treated with furosemide. However, this hydration scheme may not be sufficient for patients with normal BNP levels and/or LVEDP levels in the normal or low range, which can aggravate renal dysfunction. (2) Patients meeting the conditions of the experimental group had an equivalent increase in volume load, in which the 20 mg of furosemide provided on a hydration-level basis was relatively advantageous. Diuresis can accelerate the excretion of contrast agents and reduce the damage of the contrast agent to the renal tubules.

CIN, a complication associated with PCI, is an important component of iatrogenic renal injury. It has become a vital issue for cardiologists because it may prolong hospitalization and affect long-term prognosis. To our knowledge, the fundamental pathogenesis of CIN is ischemia and hypoxia in the renal medulla. At present, hydration is the only recognized clinical strategy to prevent CIN according to the IIA recommended level in the guidelines [7]. However, due to the limitations of hydration therapy shown in our previous study [5], special attention should be paid to the degree of hydration in clinical practice, especially for patients with heart failure. Notably, hyperhydration may increase the potential for pulmonary edema.

Furosemide administration has a renal protective effect against CIN [8]. Theoretically, furosemide administration attenuates direct toxicity through increased urine flow by enhancing contrast dilution in the renal tubule. Furosemide then inhibits tubular sodium reabsorption in the medulla. Concurrently, furosemide reduces the tubular burden and oxygen requirement, alleviating hypoxia of the ascending medullary branch. The mechanism mentioned above has been verified by the findings of a coauthor's clinical study [4]. Similarly, a clinical meta-analysis of randomized trials has found that furosemide with matched hydration using the RenalGuard system reduced the incidence of contrast-induced acute kidney injury in high-risk patients undergoing PCI or transcatheter aortic valve replacement [9]. The authors suggested that maintaining a high urine output (>300 mL/h) during the operation has a direct protective effect on the renal tubular cells. However, some studies with small sample sizes have shown that the administration of prophylactic furosemide may lead to deterioration of renal function after PCI [10]. A meta-analysis conducted in 2015 has indicated that furosemide fails to exert benefits in reducing the incidence of CIN and long-term adverse events after PCI [11]. Therefore,

Table 1: Baseline clinical characteristics of all patients.

Factors	Control group	Experimental group	*P* value
Female sex (%)	28.60%	27.43%	0.673
Age (years)	59.00 (13.00)	59.00 (12.00)	0.803
Weight (kg)	70.00 (15.00)	71.00 (15.00)	0.815
Height (cm)	170.00 (11.00)	169.00 (11.00)	0.525
Hypertension, *n* (%)	305 (57.77%)	309 (58.86%)	0.719
Diabetes, *n* (%)	141 (26.70%)	145 (27.62%)	0.739
Hyperlipidemia, *n* (%)	225 (42.61%)	240 (45.71%)	0.311
Smoking, *n* (%)	202 (38.48%)	208 (39.39%)	0.760
Statins, *n* (%)	457 (86.55%)	435 (82.86%)	0.096
ACEI/ARB, *n* (%)	215 (40.72%)	219 (41.71%)	0.743
Ejection fraction (%)	61.86 (3.03)	61.97 (2.52)	0.297
Left ventricular diameter (mm)	48.00 (6.00)	47.00 (6.00)	0.021
LVEDP (mmHg)	15.00 (9.00)	15.00 (8.00)	0.923
BNP before PCI (pg/mL)	31.60 (67.70)	30.30 (61.00)	0.677
Total input in 24 h (mL)	4250.66 ± 1006.65	4141.35 ± 1022.26	0.046
Total output in 24 h (mL)	4539.96 ± 1092.81	4521.02 ± 1838.11	0.178
Fasting blood glucose (mmol/L)	4.98 (1.56)	5.06 (1.58)	0.498
Total cholesterol (mmol/L)	4.07 (1.22)	4.08 (1.39)	0.212
Albumin (g/L)	40.90 (5.30)	41.40 (4.90)	0.090
Creatinine (umol/L)	69.45 (17.33)	69.30 (18.80)	0.772
Uric acid (umol/L)	298.00 (105.00)	304.00 (110.00)	0.701
Cystatin C (mg/L)	0.97 (0.30)	0.96 (0.33)	0.878
Macroglobulin (mg/L)	1.50 (0.72)	1.54 (0.67)	0.213
Platelets (10^9/L)	206.00 (71.00)	204.00 (68.00)	0.619
Hemoglobin (g/L)	138.00 (18.00)	138.00 (18.00)	0.684
Homocysteine (umol/L)	13.95 (11.93)	12.95 (11.93)	0.016
C-reactive protein (mg/L)	3.00 (4.58)	2.40 (3.58)	0.007
Iopromide, *n* (%)	454 (85.98%)	470 (89.52%)	0.080
Contrast agent dose (mL)	120.00 (60.00)	100.00 (60.00)	0.197
Right radial artery, *n* (%)	451 (85.90%)	457 (87.05%)	0.443
Acute myocardial infarction, *n* (%)	195 (36.93%)	186 (35.43%)	0.612
Severity of coronary artery disease, *n* (%)			
Single branch	258 (48.86%)	237 (45.14%)	0.354
Double branch	157 (29.73%)	158 (30.10%)	0.898
Three branches	113 (21.40%)	130 (24.76%)	0.196

ACEI/ARB, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers; BNP, B-type natriuretic peptide; LVEDP, left ventricular end-diastolic pressure; PCI, percutaneous coronary intervention.

Table 2: Incidence of contrast-induced nephropathy in the control and experimental groups.

Group	Patients receiving furosemide (*n*)	CIN	Incidence (%)	*P* value
Control ($n = 528$)	528	43	8.14	<0.001
Experimental ($n = 525$)	381	15	2.86	

CIN, contrast-induced nephropathy.

Table 3: Incidence of CIN in different subgroups within the experimental group.

Group	Patients treated with furosemide (*n*)	CIN	Incidence (%)	*P* value
BNP※ ($n = 78$)	78	3	3.85	
LVEDP☆ ($n = 153$)	153	4	2.61	0.95
BNP + LVEDP◎ ($n = 15$)	150	4	2.67	
Incompatible with both standards ($n = 144$)	0	4	2.78	

BNP, B-type natriuretic peptide; CIN, contrast-induced nephropathy; LVEDP, left ventricular end-diastolic pressure. Patients in the experimental group received furosemide if they showed LVEDP ≥ 15 mmHg, a post-PCI BNP ≥ 100 pg/mL, and/or a post-PCI BNP value of >150% of the pre-PCI value. ※Use of furosemide only met the BNP standard; ☆Use of furosemide only met the LVEDP standard; ◎Use of furosemide met the BNP and LVEDP standards.

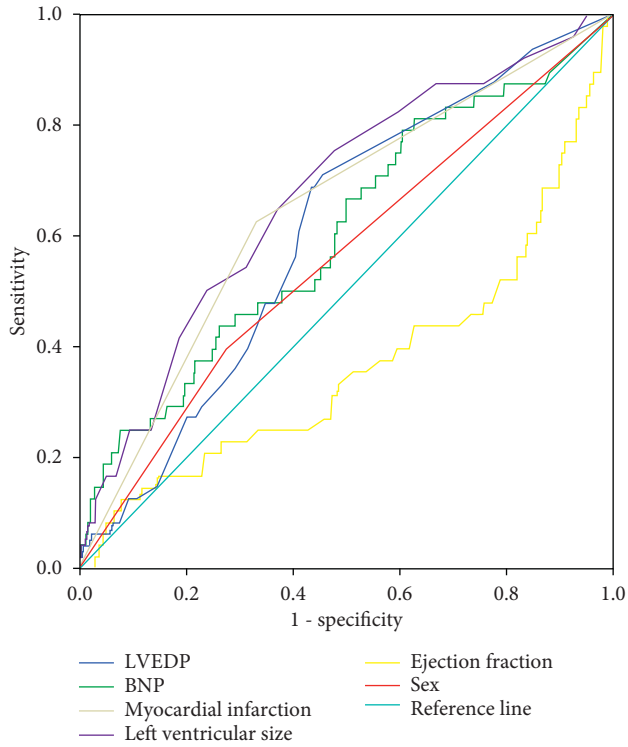

Figure 1: The area under the curve for the predictive performance of different indexes for contrast-induced nephropathy incidence in the cohort. BNP, B-type natriuretic peptide; LVEDP, left ventricular end-diastolic pressure.

according to these different views, our study aimed to identify indicators that could guide precise hydration treatment and to serve as a guide for the protective administration of furosemide to prevent CIN.

Accurate evaluation of blood volume status is the key step in optimizing hydration therapy. The LVEDP, which can accurately reflect the left ventricular preload, has been used as the evaluation index. A single-center, retrospective study has shown that LVEDP is an independent predictor of CIN (OR, 3.41; 95% CI, 2.34–4.99), and the incidence of CIN in patients with LVEDP ≥20 mmHg (19.5%) significantly increases [12]. Moreover, for every 5 mmHg increase in LVEDP, the risk of CIN increases by 1.26 times. Several studies have reported that intervention based on LVEDP can significantly reduce the incidence of CIN and major adverse events (all-cause mortality, myocardial infarction, and long-term hemodialysis) at late follow-up [13, 14]. An increase in LVEDP values beyond the normal range indicates a change in left ventricular pressure, which leads to an increase in BNP release. Stolker and Rich [15] have pointed out that BNP level is an independent predictor of increased LVEDP in elderly patients undergoing diagnostic cardiac catheterization, and a BNP level increase can increase the estimated LVEDP value [16]. Therefore, the combined monitoring of LVEDP and BNP levels to evaluate the degree of hydration can inform individualized diuretic plans to improve clinical accuracy. In particular, compared with LVEDP, BNP is relatively easy to obtain.

4.1. Study Limitations. In interpreting these data, some limitations should be considered. No long-term follow-up study was conducted on these patients, and the long-term influence of the guidance program could not be obtained.

5. Conclusion

It is crucial to evaluate the LVEDP and BNP levels to optimize the administration of furosemide during PCI. Our study showed that an LVEDP value of 15.5 mmHg and a BNP level of 116.5 pg/mL were the optimal cut-off values to predict the occurrence of CIN. The administration of furosemide after the assessment of effective blood volume can further reduce the occurrence of CIN, which indicates the effectiveness of individualized precision treatment schemes and is worthy of further promotion in clinical practice.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Disclosure

The funding sources had no roles in the study design; in the collection, analysis, or interpretation of data; in the writing of the report; or in the decision to submit the article for publication.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

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Supplementary Materials

Supplementary Table S1: Comparison of renal function between the experimental and control groups. Supplementary Table S2: Logistic regression analysis was performed to further explore the predictive value of LVEDP and BNP for CIN. CONSORT checklist: CONSORT 2010 checklist of information to include when reporting a randomized trial. . (*Supplementary Materials*)

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