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CONTRAST-INDUCED NEPHROPATHY

Prevention of Contrast-Induced Nephropathy by Central Venous Pressure–Guided Fluid Administration in Chronic Kidney Disease and Congestive Heart Failure Patients

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ABSTRACT

OBJECTIVES This study aimed to explore the hemodynamic index-guided hydration method for patients with congestive heart failure (CHF) and chronic kidney disease (CKD) to reduce the risk of contrast-induced nephropathy (CIN) and at the same time to avoid the acute heart failure.

BACKGROUND Patients at moderate or high risk for CIN should receive sufficient hydration before contrast application.

METHODS This prospective, randomized, double-blind, comparative clinical trial enrolled 264 consecutive patients with CKD and CHF undergoing coronary procedures. These patients were randomly assigned to either central venous pressure (CVP)-guided hydration group ($n = 132$) or the standard hydration group ($n = 132$). In the CVP-guided group, the hydration infusion rate was dynamically adjusted according to CVP level every hour. CIN was defined as an absolute increase in serum creatinine (SCr) >0.5 mg/dl (44.2 μmol/l) or a relative increase >25% compared with baseline SCr.

RESULTS Baseline characteristics were well-matched between the 2 groups. The total mean volume of isotonic saline administered in the CVP-guided hydration group was significantly higher than the control group (1,827 ± 497 ml vs. 1,202 ± 247 ml; $p < 0.001$). CIN occurred less frequently in CVP-guided hydration group than the control group (15.9% vs. 29.5%; $p = 0.006$). The incidences of acute heart failure during the hydration did not differ between the 2 groups (3.8% vs. 3.0%; $p = 0.500$).

CONCLUSIONS CVP-guided fluid administration can safely and effectively reduce the risk of CIN in patients with CKD and CHF. (Central Venous Pressure Guided Hydration Prevention for Contrast-Induced Nephropathy; NCT02405377) (J Am Coll Cardiol Intv 2016;9:89–96) © 2016 by the American College of Cardiology Foundation.

Incidence of contrast-induced nephropathy (CIN) is reported to be more than 20% in chronic kidney disease (CKD) complicated with congestive heart failure (CHF) (1–3), and CIN is a significant risk factor for long-term mortality and renal events after coronary angiography (4,5). Patients at moderate or high risk for CIN should receive sufficient hydration before and after application of contrast (6). Hydration

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ABBREVIATIONS AND ACRONYMS

CHF = congestive heart failure

CIN = contrast-induced nephropathy

CKD = chronic kidney disease

CVP = central venous pressure

eGFR = estimated glomerular filtration rate

LVEF = left ventricular ejection fraction

SCr = serum creatinine

is the cornerstone for prevention of CIN, because hydration could increase the renal flow, reducing the contraction of renal vessels and the formation of urinary casts (7–9). The guidelines recommend controlling the hydration rate in patients with CHF to avoid acute pulmonary edema. However, inadequate hydration markedly increases the incidence of CIN in patients with CKD (9). We expect to explore an individual hydration method for CKD-complicated CHF patients to reduce the incidence of CIN and, at the same time, to prevent acute heart failure.

METHODS

The study was a prospective, randomized, double-blind clinical trial, conducted in China from February 2014 to February 2015. This trial was registered with ClinicalTrials.gov (NCT02405377). The study was approved by the institutional review board of the Chinese People's Liberation Army General Hospital and performed in conformity with the Helsinki Declaration of 1975, as revised in 2000. The ethical committee of our institution approved the protocol. Written informed consent was provided by all patients before enrollment.

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STUDY POPULATION. We enrolled patients from February 2014 to December 2014. The principal inclusion criteria included: 1) CHF: left ventricular ejection fraction (LVEF) by echocardiography <50% and with a typical attack of congestive left heart failure in the past 1 year, which presented with paroxysmal nocturnal dyspnea or orthopnea with obvious rales or wheezes in lungs; 2) CKD: estimated glomerular filtration rate (eGFR) from 15 to 60 ml/min/ 1.73 m^2, calculated via the Modification of Diet in Renal Disease study equation (10); and 3) patients scheduled to undergo diagnostic cardiac angiography or percutaneous coronary intervention. The principal exclusion criteria included: dialysis-dependent patients; age <18 years; pregnancy; lactation; emergency cardiac catheterization (e.g., primary percutaneous coronary intervention for ST-segment elevation myocardial infarction); exposure to radiographic contrast media within the previous 7 days; acute decompensated heart failure; and cardiogenic shock. We randomly assigned eligible patients at a 1:1 ratio to either central venous pressure (CVP)-guided hydration or standard hydration protocol. Eligible patients were assigned with sealed blinded envelopes that contained a computer-generated randomization number. Patients were not told to which group they were randomly allocated. The cardiologists performing the angiogram also had no knowledge of each patient's group assignment.

PROCEDURES. We used 0.9% sodium chloride hydration in all patients. We monitored the CVP level by placing a 5-F catheter in the jugular vein and recorded initial CVP level in both groups. Patients in the CVP-guided hydration group were divided into 3 subgroups according to initial CVP level, group 1 (CVP <6 cm H_2O), group 2 (CVP 6 to 12 cm H_2O), and group 3 (CVP >12 cm H_2O). The rate of fluid administration was guided by CVP as follows: 3 ml/kg/h for group 1, 1.5 ml/kg/h for group 2, and 1 ml/kg/h for group 3. The intravenous infusion rate was dynamically adjusted according to the level of CVP per hour during hydration. If the CVP of patients experienced a rise among the groups, (for example, if CVP increased from 8 cm H_2O to 13 cm H_2O, the patient would change from group 2 to group 3), this would necessitate a reduction in the intravenous infusion rate from 1.5 ml/kg/h to 1 ml/kg/h. If the patient's CVP increased from 8 cm H_2O to 12 cm H_2O, the fluid rate remained 1.5 ml/kg/h. The control group was hydrated at the rate of 1 ml/kg/h. Hydration continued from 6 h before the procedure to 12 h post-procedure, thus both study groups received intravenous fluids for the same duration but at different rates. All study participants received iodixanol (320 mg I/ml; Visipaque, GE Healthcare, Chalfont St. Giles, United Kingdom) as the contrast medium.

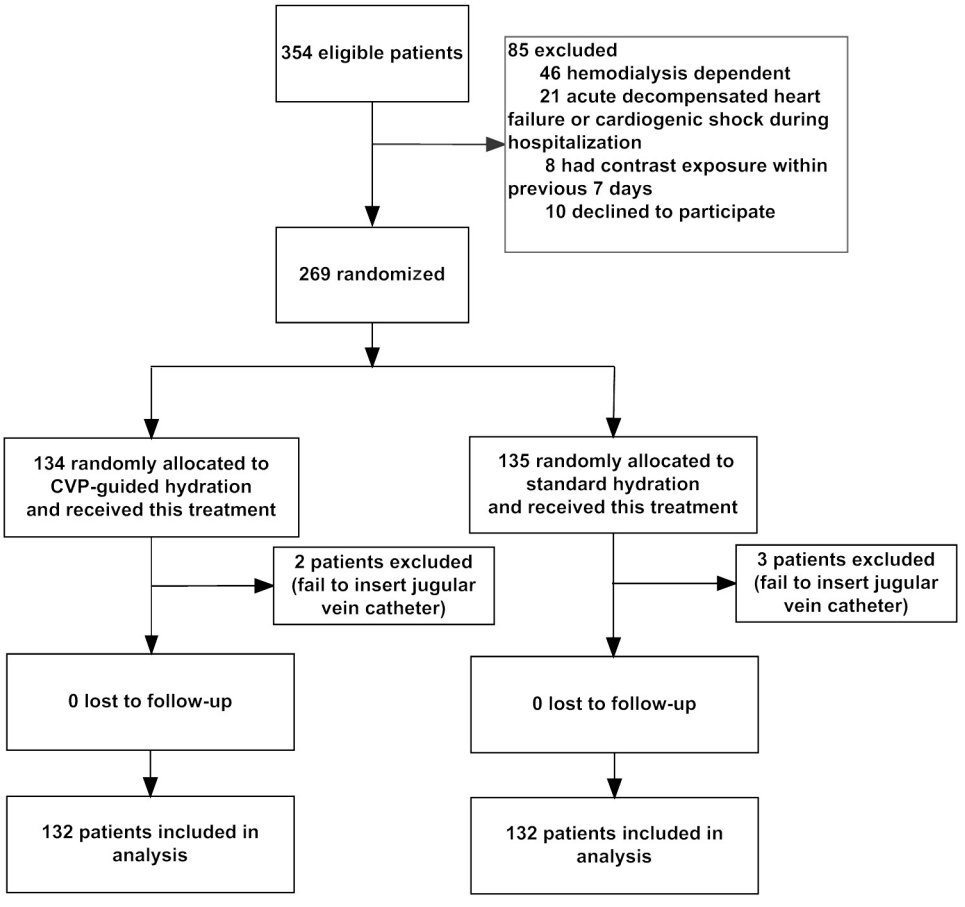
ENDPOINTS AND DEFINITION. The primary endpoint of the study was CIN, defined as the peak increase in serum creatinine (SCr) concentration either ≥25% or ≥0.5 mg/dl (44.2 μmol/l) over baseline during the first 72 h post-procedure, and we further analyzed the proportion of patients with a peak SCr increase ≥50% or ≥1.0 mg/dl (88.4 μmol/l) over baseline in the initial 72 h post-procedure. Urine output, SCr, blood urea nitrogen, and serum electrolytes were also evaluated at baseline, the day of coronary angiography and each day for the following 3 days and at hospital discharge for assessment of acute kidney injury severity and indication of dialysis. Secondary endpoints were major post-procedure adverse clinical events including acute pulmonary edema, myocardial infarction, all-cause death, and CIN requiring renal replacement therapy. Myocardial infarction was defined as a creatine kinase-myocardial band enzyme elevation 3 times the upper normal value with or without new Q waves on the electrocardiogram. The risk score of CIN was assessed on the basis of the patients' clinical and laboratory conditions (1,3). Each

patient was contacted every week after administration of the contrast medium, investigated as to whether dialysis or main cardiovascular events had occurred within 3 months after the coronary procedure, and any adverse events were recorded. All adverse clinical events, as well as study endpoints, were judged by the independent event committee, whose members were masked to treatment assignment.

STATISTICAL ANALYSIS. Clinical characteristics and laboratory features of our study population were analyzed. Data were expressed as mean ± SD for continuous variables and as percentages for discrete variables. Continuous variables were compared between the groups using the Student *t* test (for normal distribution) or Mann-Whitney rank sum test (for nonnormal distribution). Categorical variables are presented as numbers and percentages and were compared with chi-square or Fisher exact tests when there were <5 values in a given cell. We compared the occurrence of major adverse events with the Kaplan-Meier method using the log-rank test. All statistical tests used a 2-sided test, and $p < 0.05$ indicated statistical significance. All statistical analyses were performed using SPSS statistical software (version 18.0, SPSS, Chicago, Illinois).

We calculated the necessary sample size before carrying on the research. According to authoritative literature, the incidence of CIN in patients who meet inclusion criteria is about 25% (1,3). Our preliminary experiment showed that CVP-guided hydration could reduce the incidence of CIN at least by 40%. On the basis of these assumptions, chi-square analysis suggested that 210 patients would be needed to detect a statistically significant difference, with 80% power and a 2-sided α of 0.05. If the loss to follow-up was 10%, 120 patients were needed for each group.

FIGURE 1 Flow Diagram of the Trial



From 354 eligible patients, 269 were randomized to receive CVP-guided hydration (n = 134) or standard hydration (n = 135), 264 patients were followed up and completed the study. CVP = central venous pressure.

RESULTS

BASELINE CHARACTERISTICS AND PROCEDURES. Of 354 consecutive eligible patients, 269 were enrolled and randomly allocated to the CVP-guided treatment group (n = 134) or the control group (n = 135). Of these, 2 patients in the CVP-guided treatment group and 3 patients in the control group were excluded due to failure of jugular vein catheterization, and 264 patients who were randomly assigned to either the CVP-guided treatment group (n = 132) or the control group (n = 132) completed the study and were included in the primary analysis (Figure 1). We followed up study patients from February 2014 to February 2015. Baseline characteristics and procedures for study population are shown in Table 1. The mean age of the cohort was 64 years, with 25% women and 47% patients with diabetes. Baseline characteristics were well matched between the 2 groups. The mean eGFR (median [interquartile range]) was similar between the 2 groups (36 [23 to 48] ml/min/1.73 m^2 vs. 39 [26 to 52] ml/min/1.73 m^2; p = 0.224). Predicted risk of CIN (24 ± 7% vs. 25 ± 8%, p = 0.543) were comparable between the 2 groups. The distributions of CVP were well matched between the 2 groups (p = 0.780). The total mean volume of saline administered in the CVP-guided group was significantly higher than in the control group (1,827 ± 497 ml vs. 1,202 ± 247 ml; p < 0.001) (Figure 2); meanwhile, patients in the CVP-guided hydration group had a higher volume of urine output than the control group (1,461 ± 453 ml vs. 806 ± 228 ml; p < 0.001). In a different phase of hydration, the volumes of isotonic saline administered in the CVP-guided hydration group were all significantly higher than in the control group (719 ± 331 ml vs. 385 ± 71 ml in the pre-procedural phase, p < 0.001; 130 ± 66 ml vs. 86 ± 45 ml in intraprocedural phase, p = 0.004; 978 ± 403 ml vs. 731 ± 181 ml in post-procedural phase, p < 0.001).

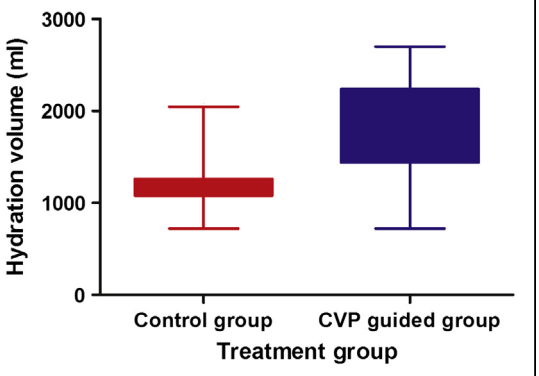
TABLE 1 Basic Characteristics of Study Patients

	CVP-Guided Hydration Group (n = 132)	Control Group (n = 132)	p Value
Age, yrs	64 ± 10	63 ± 12	0.524
Sex, male/female	101/31	96/36	0.572
Weight, kg	73 ± 12	70 ± 13	0.069
Height, cm	167 ± 7	166 ± 7	0.130
CVP, cm H_2O	10 ± 4	9 ± 4	0.185
Distribution of CVP			0.780
CVP <6 cm H_2O	28 (21.2)	24 (18.2)	
CVP 6-12 cm H_2O	69 (52.3)	74 (56.1)	
CVP >12 cm H_2O	35 (26.5)	34 (25.8)	
Clinical profile			
Acute coronary syndrome	107 (81.1)	108 (81.8)	1.000
Hypercholesterolemia	25 (18.9)	25 (18.9)	1.000
Hypertension	112 (84.8)	103 (78.0)	0.267
Diabetes mellitus	67 (50.8)	58 (43.9)	0.325
History of smoking	67 (50.8)	61 (46.2)	0.538
SBP, mm Hg	136 ± 19	133 ± 25	0.168
DBP, mm Hg	76 ± 13	74 ± 14	0.366
SBP <100 mm Hg	15 (11.4)	16 (12.1)	0.500
Heart rate, beats/min	77 ± 11	79 ± 13	0.237
Heart rate >100 beats/min	13 (9.8)	16 (12.1)	0.347
Laboratory data			
LDL-c, mmol/l	2.48 ± 0.87	2.39 ± 0.95	0.432
TC, mmol/l	4.07 ± 1.17	4.14 ± 1.09	0.621
ALT, U/l	22 ± 18	27 ± 20	0.018
Serum glucose, mmol/l	7.8 ± 5.0	8.5 ± 4.0	0.223
Hemoglobin, g/l	117 ± 24	122 ± 24	0.096
Count of white blood cell, $\times 10^9$/l	7.72 ± 2.89	8.36 ± 3.67	0.118
Ejection fraction, %	40 ± 8	39 ± 8	0.160
Estimated GFR, ml/min/1.73 m^2	36 (23-48)	39 (26-52)	0.224
Serum creatinine, μmol/l	155 (130-271)	147 (113-256)	0.147
Predictive CIN risk score	13 ± 4	13 ± 4	0.429
Predictive risk of CIN, %	24 ± 7	25 ± 8	0.543
Medication			
Loop diuretics	65 (49.2)	70 (53.0)	0.622
Statins	132 (100)	132 (100)	1.000
N-acetylcysteine	113 (85.6)	109 (82.6)	0.614
ACE-i/ARBs	83 (62.9)	71 (53.8)	0.170
PCI	113 (85.6)	119 (90.2)	0.346
Hydration volume, ml	1,827 ± 497	1,202 ± 247	<0.001
Contrast medium volume, ml	161 ± 67	171 ± 67	0.205

Values are mean ± SD or n (%), or median (interquartile range), except for estimated GFR and serum creatinine: median (interquartile range).

ACE-i/ARB = angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; ALT = alanine aminotransferase; CIN = contrast-induced nephropathy; CVP = central venous pressure; DBP = diastolic blood pressure; GFR = glomerular filtration rate; LDL-c = low-density lipoprotein cholesterol; PCI = percutaneous coronary intervention; SBP = systolic blood pressure; TC = total cholesterol.

FIGURE 2 Hydration Volumes of Normal Saline Administered in Each Group



The **box for each group** represents the 25th percentile to the 75th percentile of the data. The **whiskers** start from minimum data point to the maximum data point. The mean of hydration volumes was significantly higher in the CVP-guided hydration group than in the control group. CVP = central venous pressure.

INCIDENCE OF CIN. The overall incidence of CIN for enrolled high-risk patients was 22.7% (60 of 264): it was 15.9% (21 of 132) in the CVP-guided hydration group versus 29.5% (39 of 132) in the control group ($p = 0.006$). The absolute risk difference was 13.6% (95% confidence interval: 3.6% to 23.7%). The proportion of patients with SCr increasing >0.3 mg/dl and >50% from baseline were both significantly lower in the CVP-guided hydration group than in the control group (3.79% vs. 9.85%; $p = 0.042$ in SCr >50%; 19.7% vs. 34.8%; $p = 0.004$ in SCr >0.3 mg/dl) (Table 2). Differences of outcomes in CIN between study groups were probably due to more aggressive volume expansion guided by CVP. The whole study cohort was divided into 3 groups on the basis of the volume of isotonic saline received. The volume of fluid in each group was: group 1 (500 to 1,000 ml), group 2 (1,000 to 1,500 ml), and group 3 (>1,500 ml). The corresponding rates of CIN were 37.9% (11 of 29) for group 1, 31.3% (41 of 131) for group 2, and 7.7% (8 of 104) for group 3. There was a strong negative correlation between hydration volume and the increase of SCr ($r = -0.21$, $p < 0.001$) (Figure 3). We analyzed the relationship between CIN and subgroups defined by level of LVEF and CVP. The patients with worse LVEF (<40%) got more benefit for CIN prevention from the hydration guided by CVP dynamic monitoring (17.5% vs. 33.3%; $p = 0.031$), and the patients with the lowest CVP level (<6 cm H_2O) received the greatest benefits from the CVP-guided hydration (10.7% vs. 37.5%; $p = 0.045$), as shown in Table 3.

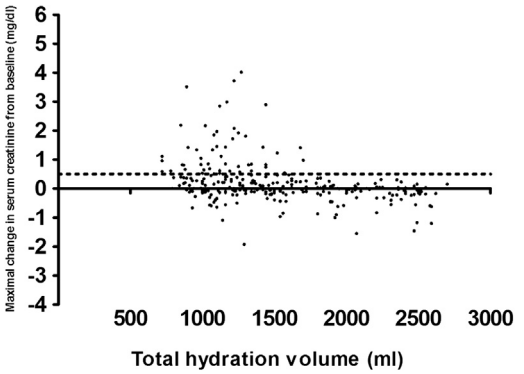
ADVERSE EVENTS. During the hospital stay and out to 90 days after the procedure, myocardial infarction incidence was much lower in the CVP-guided treatment group than in the control group (4 [3.0%] vs. 13 [9.8%]; $p = 0.019$], and renal replacement therapy was also significantly lower in the CVP-guided treatment group than in the control group (4 [3.0%] vs. 13 [9.8%]; $p = 0.019$). Although the volume of the isotonic saline received was much higher in the CVP-guided group than the control group, the aggressive volume expansion was terminated at a similarly low rate because of acute pulmonary edema (5 [3.8%] vs. 4 [3.0%]; $p = 0.500$), and 52 patients in the CVP-guided hydration group had a reduction in the infusion rate due to an obvious increase of CVP. The incidence of 90-day acute heart failure did not differ between the 2 groups (9 [6.8%] vs. 10 [7.6%]; $p = 0.500$). Nine patients who had an episode of acute pulmonary edema during hospitalization had worse LVEF (35 ± 6% vs. 40 ± 8%; $p = 0.061$), higher initial CVP level (15 ± 1 cm H_2O vs. 9 ± 4 cm H_2O; $p < 0.001$), higher heart rate (94 ± 20 vs. 78 ± 12; $p < 0.001$), lower eGFR (24 ± 13 ml/min/1.73 m^2 vs. 40 ± 21 ml/min/1.73 m^2; $p = 0.019$), and received a higher volume of contrast medium (217 ± 75 ml vs. 164 ± 66 ml; $p = 0.019$) compared with other study patients who did not experience acute pulmonary edema, and they received a lower hydration volume compared with other study patients (1,220 ± 250 ml vs. 1,525 ± 505 ml; $p = 0.073$); however, a lower hydration volume still did not prevent acute pulmonary edema. Ninety days after the procedure, cumulative major adverse events were reported in 8.3% (11 of 132) of patients in the CVP-guided hydration group compared with 20.5% (27 of 132) in the control group ($p = 0.004$) (Table 4). Figure 4 shows the occurrence of major adverse events during the 90-day follow-up by treatment group (p for log-rank = 0.005). We also reported the rate of major adverse events by CIN. In

TABLE 2 Incidence of CIN in Study Patients

Definition of CIN	CVP-Guided Hydration Group (n = 132)	Control Group (n = 132)	Absolute Difference (95% CI)	p Value
SCr >50% ↑	5 (3.79)	13 (9.85)	3.1 (0.0–12.1)	0.042
SCr >25% ↑	19 (14.4)	32 (24.2)	9.8 (0.3–19.4)	0.030
SCr >0.5 mg/dl ↑	18 (13.6)	35 (26.5)	4.9 (3.3–22.5)	0.007
SCr >0.3 mg/dl ↑	26 (19.7)	46 (34.8)	15.2 (4.5–25.8)	0.004
Incidence of CIN	21 (15.9)	39 (29.5)	13.6 (3.6–23.7)	0.006

Values are n (%).
CI = confidence Interval; SCr = serum creatinine; other abbreviations as in Table 1.

FIGURE 3 Relationship Between Absolute SCr Change and Total Hydration Volume



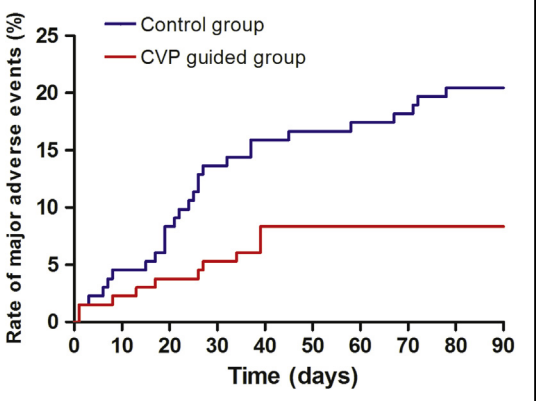
The **dotted line** indicates the 0.5 mg/dl of serum creatinine (SCr) increase from baseline used for the definition of contrast-induced nephropathy. There is a strong negative correlation between hydration volume and increase of SCr.

TABLE 3 Incidence of CIN in Study Patients by Subgroups

	CVP-Guided Hydration Group	Control Group	Absolute Difference, % (95% CI)	p Value
LVEF				
40%–50%	10/69 (14.5)	17/66 (25.8)	11.3 (−2.3 to 24.8)	0.077
<40%	11/63 (17.5)	22/66 (33.3)	15.8 (0.9 to 30.9)	0.031
CVP				
<6 cm H_2O	3/28 (10.7)	9/24 (37.5)	26.8 (4.0 to 49.6)	0.045
6–12 cm H_2O	8/69 (11.6)	20/74 (27.0)	15.4 (2.5 to 28.4)	0.022
>12 cm H_2O	10/35 (28.5)	10/34 (29.4)	0.8 (−21.3 to 23.0)	0.939

Values are n/N (%).
LVEF = left ventricular ejection fraction; other abbreviations as in Tables 1 and 2.

FIGURE 4 Cumulative Major Adverse Events at 90 Days for the CVP-Guided Hydration Group Versus Control Group



The graph shows the 3-month major adverse events, defined as a composite of all-cause mortality, myocardial infarction, and acute renal failure requiring dialysis in each group. CVP = central venous pressure.

patients with CIN, the rate of major adverse events was 38% (23 of 60), and patients who developed CIN had a higher rate of all-cause mortality than those who did not develop CIN (p = 0.017).

DISCUSSION

The study population was at a high risk of CIN, and the incidence of contrast-induced acute kidney injury in the control group was similar to the expected incidence according to the CIN risk score (1,3). This trial found that CVP-guided hydration resulted in a significant reduction of 46% in the primary endpoint of CIN and a significant reduction of 59% in major adverse clinical events compared with standard treatment. Our study revealed that CIN could be further reduced with CVP-guided hydration.

The mechanisms of these favorable treatment effects are probably multifactorial. Volume expansion with saline reduced CIN by reducing the contrast medium concentration, suppression of the renin-angiotensin-aldosterone system, and down-regulation of tubuloglomerular feedback, by dilution of the contrast medium within the tubular lumen (11–14). We found that the CVP in some of the CHF patients were at relatively low levels, and CHF patients were often accompanied by renal perfusion defects (15); sufficient hydration could maintain a constant intravascular volume and improve the renal perfusion for prevention CIN (16). In patients with CKD, hydration is usually performed at a low rate because of the fear of overhydration and pulmonary edema, particularly in patients with impaired left ventricular function. It should be emphasized that individual and adequate hydration could prevent CIN (17–19). A new concept is emerging for CIN prevention: hydration volume should be commensurate to the risk of CIN. In support of this idea, we noted that CVP-guided aggressive fluid administration increased urine flow rates by maintenance of constant intravascular volume and further reduced the risk of CIN.

As noted in current guidelines, no clear evidence exists to guide the choice of the optimal rate of fluid administration. We sought to develop a practical and feasible hydration protocol that can benefit high CIN risk patients. There have been some clinical trials on optimal fluid administration for CIN prevention (17,20–23). The MYTHOS (Induced Diuresis With Matched Hydration Compared to Standard Hydration for Contrast Induced Nephropathy Prevention) trial has revealed that diuresis with matched hydration could reduce the incidence of CIN and is associated with improved in-hospital outcome (20). RenalGuard System-guided hydration significantly decreased the incidence of CIN in high-risk patients for CIN (21). The POSEIDON (Prevention of Contrast Renal Injury With Different Hydration Strategies) trial also showed left ventricular end-diastolic pressure-guided hydration was safe and effective in preventing CIN and

TABLE 4 Ninety-Day Main Adverse Events in Study Patients

	CVP-Guided Hydration Group (n = 132)	Control Group (n = 132)	Absolute Difference, % (95% CI)	p Value
All-cause mortality	3 (2.3)	5 (3.8)	1.5 (−2.7 to 5.7)	0.722
Myocardial infarction	4 (3.0)	13 (9.8)	6.8 (0.9 to 12.7)	0.019
Renal replacement therapy	4 (3.0)	13 (9.8)	6.8 (0.9 to 12.7)	0.019
Cumulative major adverse events	11 (8.3)	27 (20.5)	12.1 (3.7 to 20.5)	0.004

Values are n (%).
Abbreviations as in Tables 1 and 2.

improving the outcome (17). In our study, we applied hemodynamic CVP instead of left ventricular end-diastolic pressure to assess volume status and the ability to tolerate high rates of fluid administration for these CHF patients and had made similar conclusions.

It cannot be ignored that the hydration process also could further increase cardiac pre-load (9,15). CVP provides a safe and accurate assessment of intravascular volume status. We adjusted the velocity of hydration to ensure sufficient hydration without obvious increasing the volume load, and the aggressive volume expansion was terminated at a similarly low rate due to pulmonary edema (5 [3.8%] vs. 4 [3.0%]; $p = 0.500$); 52 patients in CVP-guided hydration group had reduction in infusion rate due to an obvious increase of CVP, all of which prove the safety of the CVP-guided hydration procedure.

STUDY LIMITATIONS. First, this trial was a single-center study. Second, the physicians undertaking the hydration procedure were not blinded to the treatment groups, which might have influenced our results. Last, the strategy of aggressive volume expansion was not suitable for all high-risk patients, especially those with acute decompensated heart failure. The conclusion needs to be tested in more patients in multiple centers.

CONCLUSIONS

CVP-guided fluid administration can safely and effectively reduce the risk of CIN in patients with CKD and CHF and substantially reduce composite major adverse events for these high-risk patients.

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PERSPECTIVES

WHAT IS KNOWN? Despite general agreement on hydration being the cornerstone for prevention of CIN and strong recommendation of all guidelines, CHF patients are not receiving sufficient hydration in clinical practice because of fear of increased cardiac pre-load and pulmonary edema during hydration. CVP could objectively reflect change of intravascular volume status and the body's tolerance to aggressive hydration. It is unclear whether CVP-guided fluid administration could decrease the risk of CIN for patients with CHF and CKD.

WHAT IS NEW? This study shows aggressive volume expansion guided by CVP (monitoring during hydration to adjust the rate of saline administration dynamically) resulted in a significant 46% reduction in the primary endpoint of CIN compared with standard treatment; meanwhile, acute pulmonary edema during hydration did not differ between the CVP-guided hydration group and the control group. CVP-guided vigorous fluid administration could avoid fluid overload and effectively reduce the risk of CIN in patients with CKD and CHF.

WHAT IS NEXT? It is still not clear that a strategy of hemodynamic index–guided hydration is suitable for those patients undergoing urgent angiography. In addition, further clinical trials are required to determine the optimal hydration dose for CHF patients.

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