Original Article

Furosemide's Preventative Role in Contrast-Induced Nephropathy among ST-Segment Elevation Myocardial Infarction Patients undergoing Percutaneous Coronary Intervention

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Abstract

Background: ST-elevation myocardial infarction (STEMI) is regarded as a risk factor for contrast-induced nephropathy (CIN). Numerous studies have explored preventive measures for CIN such as the use of diuretics. However, the specific impact of diuretics in averting nephropathy remains uncertain. We investigated whether post-treatment with furosemide decreases CIN risk.

Materials and Methods: We designed a clinical trial and included 234 patients with STEMI undergoing primary percutaneous coronary intervention (PCI). Patients were divided into two groups: Group I (furosemide 0.5 mg/kg) and Group II (control). Serum creatinine and blood urea nitrogen (BUN) were measured pre- and post-surgery. CIN was described as more than either 25% or 0.5 mg/dL induction in serum creatinine from the baseline in 24 h and 72 h post-PCI.

Results: CIN occurred in 33 patients (14.1%) in 24 h and 35 patients (14.9%) in 72 h. CIN incidence was not significantly different between both groups. However, BUN was significantly higher in the furosemide group ($P < 0.05$). Most patients in the furosemide group were men and smokers compared to the control group.

Conclusion: A combination of low-dose furosemide plus standard hydration was not correlated with lower CIN incidence in STEMI patients who are candidates for primary PCI compared to standard hydration only. Further studies with a larger sample size in the future are needed to better understand the effects of this combination.

Keywords: Coronary artery disease, diuretics, kidney diseases, myocardial infarction

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Introduction

ST-elevation myocardial infarction (STEMI) is the most common reason for death and morbidity. The decrease in myocardial damage is the fulcrum of treatment for STEMI and is best achieved by early reperfusion via emergency percutaneous coronary intervention (PCI).[1] Cardiovascular complications post-emergency PCI with STEMI are probably lethal, thereby leading to the impairment of clinical prediction.[2] A post-myocardial infarction cardiac complication is known as contrast-induced nephropathy (CIN), which refers to acute renal damage occurring within

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48 h of a contrast agent usage in up to 15% of candidates undergoing coronary angiography.[3-5] Acute myocardial infarction (AMI) patients cured with primary PCI are at a higher risk of CIN in comparison to patients who are candidates for elective PCI.[6] The recently proved criterion for CIN is serum creatinine (Cr) levels of more than 25% of baseline, or more than 0.5 mg/dL, within 48 h of a contrast agent usage.[7,8] With the vast application of contrast agents, CIN incidence is escalating. CIN can result in higher complex clinical course, enhanced morbidity, prolonged hospitalization, higher death, and enhanced remedial expenses.[9] In subjects with mixed risk factors for CIN, it may even lead to lasting kidney injury and further dialysis therapy.[10] So, the probability of CIN occurrence is of note among cardiologists, and so attempts have been made for its suppression.

The recently proved prohibitory estimates for CIN include prophylactic hydration as well as a hypo- or isotonic contrast agent usage.[11] Furosemide has been introduced as a powerful and direct loop diuretic and kidney vasodilator, whose combination with hydration remedy is accepted for urine output increase as well as fluid overload suppression.[12] It is to be noted that, besides urine flow induction, the latter also enhances the rates of tubular flow, leads to considerable renal contrast tubular dilution, diminishes the renal metabolic workload, and afterward, lessens oxygen requirement along with ischemic damage prevention or amelioration.[13,14] Hypothetically, furosemide should have the ability to diminish CIN incidence post-radiologic processes. Nonetheless, earlier clinical investigations have shown conflicting findings on the advantage of supplemental furosemide therapy before the procedure in addition to saline hydration for CIN prevention.[15] A current study proposed that in comparison to standard hydration, induction of diuresis via furosemide with matched saline hydration dramatically lessened CIN risk in patients who were candidates for coronary procedures.[14]

Inconsistently, other researchers found a correlation of furosemide with the enhanced risk for CIN post-exposure to radiologic contrast agents.[16,17] The undesirable impact of such diuretics was also shown in more studies,[18] which only reported two small-scale investigations in terms of furosemide therapy. Other studies revealed that furosemide along with matched hydration enhanced high urine output and maintained intravascular volume, which may diminish CIN risk in chronic kidney disease patients undergoing PCI.[19] Moreover, it has been resulted that a combination of furosemide with standard hydration remedy may lessen CIN incidence and major cardiovascular events following PCI.[20] Regarding the recent contradictory proof of the clinical efficiency of furosemide for hindering CIN, we design a clinical trial study to illustrate the true impact of post-procedural furosemide strategy plus saline hydration with the aim of CIN suppression after cardiologic procedures.

Materials and Methods

Patients

A total of 234 STEMI candidates who underwent angiography and PCI at Ahvaz Golestan Hospital were prospectively recruited and screened for eligibility from 1,402 to 1,403 with a diagnosis of STEMI. Patients who were admitted with the diagnosis of STEMI and were candidates for angiography and PCI, and those with clinically complete information and consent to participate were included in the study. Exclusion criteria were patients who had heart failure or end-stage renal disease, and subjects with missing clinical data or written informed consent. The Ethics Committee of Ahvaz Jundishapur University of Medical Sciences approved the research (IR. AJUMS. HGOLESTAN.REC.1401.090) and all candidates provided their informed consent.

Study design, drug treatment, and diagnostic criteria

In this clinical trial study, eligible research candidates were randomly divided into either the control or furosemide group (n = 117 patients in each group). Before primary PCI, 5 mL of blood samples were collected from all patients to measure laboratory parameters for renal function including serum blood urea nitrogen (BUN) and Cr. For patients who were in the furosemide group, a single dose of furosemide (0.5 mg/kg over 1 h) was administrated after the surgery. Subjects in both furosemide and control groups continued to receive 1 mg/kg/h normal saline for standard hydration in the 24 h after primary PCI. Next, 24 and 72 h after the surgery, serum BUN along with serum Cr were repeated. Moreover, glomerular filtration rate (GFR) was calculated based on the following formula for each patient: GFR (mL·min $-1 \cdot 1.73$ m $^{-2}$) = 186 × serum Cr (μmol/l –1.154) × age (years–0.203) (×0.742, if female). CIN was introduced as induction of ≥25% or ≥0.5 mg/dL in comparison to pre-procedure serum Cr later than primary PCI. Finally, pre- and postoperative changes in serum Cr, serum BUN, as well as GFR were compared within each group. In the end, risk factors, including age, hypertension, dyslipidemia, diabetes mellitus and chronic kidney disease, smoking, and ischemic heart disease were compared in patients in both groups.

PCI

The PCI procedure was performed by an experienced cardiologist with radial or femoral arterial access using an iso-osmolar and non-ionic contrast medium. After passing the wire, the sheet was fixed with the appropriate size based on gender, BSA, and access placement. The catheter was inserted across the aortic cusps using a guidewire. When the guidewire was withdrawn, a catheter was engaged in the coronary artery ostium. The injection was performed using a dye and the narrowed vessel was then diagnosed. If necessary, angioplasty was performed using a balloon or appropriate stent.

Statistical analysis

The measurement data are presented as mean ± standard deviation and categorical data are shown as percentages. The chi-square test or Fisher's exact test was used to compare categorical data and CIN incidence. Independent *t*-test was used to compare continuous variables in furosemide and control groups. To consider the changes in laboratory parameters in furosemide and control groups in three investigated times, repeated measurement analysis of variance (ANOVA) was used. All statistical calculations in this research, including the *P* value, were conducted using the SPSS version 24.0 software. A *P* value less than 0.05 was considered statistically significant.

Results

Out of 234 patients who were investigated in this study, 117 patients were in the control group and 117 patients in the furosemide group. Clinical information of patients, such as age, sex, smoking status, and the presence of underlying disease, diabetes, hypertension, dyslipidemia, chronic kidney disease, and ischemic heart disease, baseline laboratory data, eGFR (mL/min/1.73), BUN (mmol/L), and serum Cr (μmol/L) compared between the two groups. There were no significant differences in any of these variables between the two groups [Table 1]. In the furosemide group, most patients were male (82.9% compared to 65% in the control group, $P < 0.05$), and smokers (47.9% compared to 29.9%, $P < 0.05$). Hypertension and diabetes are the most prominent indicators among risk factors for the occurrence of nephropathy among both groups [Table 1].

In 24 h postoperative, 31 (14.1%) patients were CIN (16 [13.7%] in the furosemide group and 17 [14.5%] in the control group). No significant difference was noted in terms of incidence of CIN (*P* value = 0.851). After 72 h, the number of CIN was 35 (15.0%) (20 [17.1%] in the furosemide group and 50 [12.8%] in the control group), no significant difference was observed at this time either. Laboratory parameters, eGFR (mL/min/1.73m^2), BUN (mg/dL), and serum Cr (mg/dL) compared between two groups in 24 and 72 h later, the results show that BUN (mg/dL) was significantly higher in the furosemide group at 24 (*P* value = 0.048) and 72 h later (0.008) [Table 2].

The results of repeated measurement analysis to consider the changes in laboratory parameters, eGFR (mL/min/1.73), BUN (mg/dL), and serum Cr (mg/dL) in both groups at three investigations times show that within-group changes were significant for eGFR (mL/min/1.73m^2) (*P* value = 0.008), BUN (mg/dL) (*P* value < 0.001), and serum Cr (mg/dL) (*P* value < 0.001). Between-group changes were significant just for BUN (mg/dL) (*P* value = 0.021). It means that these laboratory parameters change over time in both groups. The changes in eGFR (mL/min/1.73m^2) and serum Cr (mg/dL) were similar in both groups but the changes in BUN (mg/dL) in the furosemide group were more intense [Table 3]. These changes are shown in Figure 1.

Discussion

CIN occurrence in STEMI subjects who underwent primary PCI results in a detrimental clinical consequence such as higher in-hospital incidents, more venture of late cardiac incidents, and death in both subjects with ordinary kidney action along with hemodynamic inconsistency and kidney function destruction in patients with permanent chronic kidney disorder.[21] Regular hydration is known as an efficient approach for prohibiting CIN as suggested as a top recommendation in the guidelines of the European Society of Cardiology in 2018.[22,23] Furosemide may represent certain positive outcomes when it is correlated with hydration.[24] However, our results

Table 1: Baseline clinical characteristics of patients

	Population (*n*=234)	Furosemide (*n*=117)	Control (*n*=117)	*P*-value
Age (years)	56.33±10.95	55.87±10.28	56.79±11.61	0.520
Male sex, *n* %	173 (73.9)	97 (82.9)	76 (65.0)	0.002
Smoker, *n* %	91 (38.9)	56 (47.9)	35 (29.9)	0.005
Underlying disease, *n* %				
Diabetes	81 (34.6)	35 (29.9)	46 (39.3)	0.131
Hypertension	112 (47.9)	58 (49.6)	54 (46.2)	0.601
Dyslipidemia	44 (18.8)	19 (16.2)	25 (21.4)	0.315
Chronic kidney disease	13 (5.6)	5 (4.3)	8 (6.8)	0.392
Heart disease	47 (20.1)	22 (18.8)	25 (21.4)	0.624
Baseline laboratory data				
eGFR (mL/min/1.73 m^2)	87.13±33.73	85.16±30.94	89.10±36.34	0.372
Blood urea nitrogen (mg/dL)	17.80±6.90	18.40±7.56	17.21±6.15	0.186
Serum creatinine (mg/dL)	1.10±0.31	1.12±0.30	1.09±0.32	0.474

GFR: glomerular filtration rate. *P*<0.05 was significant

Table 2: Postoperative laboratory parameters in furosemide and control groups

	Population (*n*=234)	Furosemide (*n*=117)	Control (*n*=117)	*P*-value
24 hours postoperative				
CIN	33 (14.1%)	16 (13.7%)	17 (14.5%)	0.851
eGFR (mL/min/1.73 m^2)	84.41±33.22	82.91±28.65	85.90±37.30	0.492
Blood urea nitrogen (mg/dl)	19.28±7.70	20.27±8.07	18.28±7.21	0.048
Serum creatinine (mg/dl)	1.16±0.35	1.17±0.33	1.14±0.37	0.501
72 hours postoperative				
CIN	35 (15.0%)	20 (17.1%)	15 (12.8%)	0.359
eGFR (mL/min/1.73 m^2)	83.29±32.09	80.63±29.03	85.98±34.84	0.203
Blood urea nitrogen (mg/dL)	20.93±9.48	22.58±10.20	19.28±8.42	0.008
Serum creatinine (mg/dL)	1.16±0.33	1.20±0.32	1.13±0.34	0.129

GFR: glomerular filtration rate; CIN: contrast-induced nephropathy. *P*<0.05 was significant

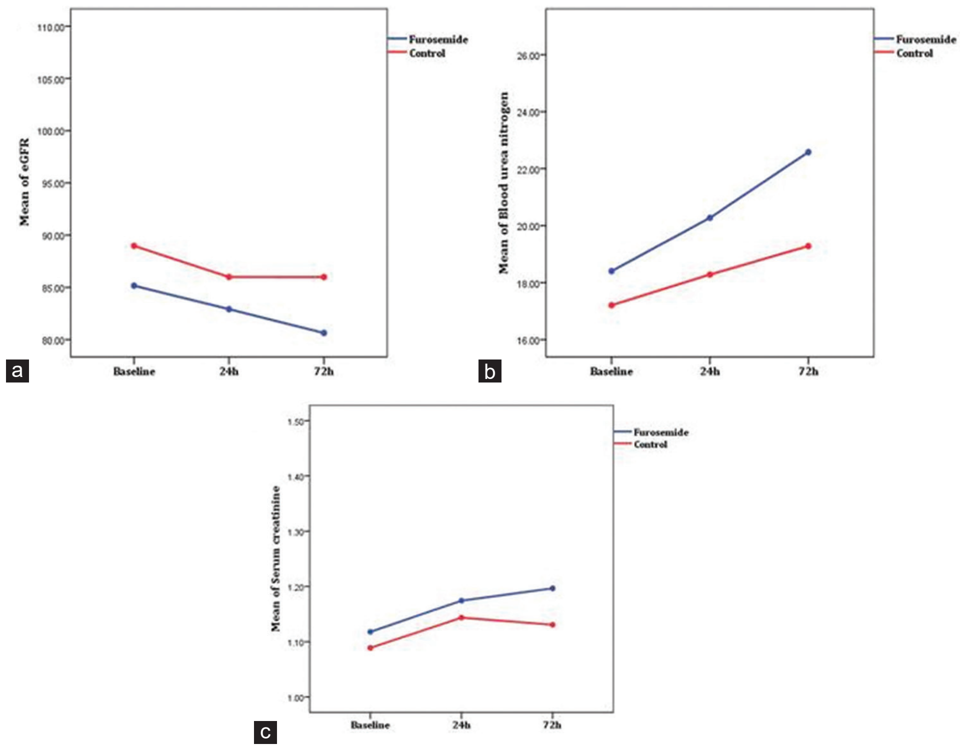

Figure 1: Changes in laboratory parameters in furosemide and control groups over time. The results of repeated measurement analysis to consider the changes in laboratory parameters in both groups at three investigation times show that within group, changes in (a) eGFR (mL/min/1.73m^2), (b) blood urea nitrogen (mg/dL), and (c) serum creatinine (mg/dL) were significant (P value < 0.001). Between group changes were just significant for blood urea nitrogen but not for eGFR and serum creatinine. It means that these laboratory parameters change over time in both groups. The changes in eGFR and serum creatinine were similar in both groups but those in blood urea nitrogen in the furosemide group were more intense

Table 3: Repeated measurement analysis for changes in laboratory parameters in furosemide and control groups

Laboratory Parameters	Investigation Times	SS	DF	MS	*F*	*P*
eGFR (mL/min/1.73 m^2)	Within group	1728.56	2	864.28	4.85	0.008
	Group × time	157.61	2	78.81	0.44	0.643
	Error	82367.30	462	178.28		
	Between-group	2912.40	1	2912.40	1.00	0.319
Blood urea nitrogen (mg/dL)	Within group	1146.18	2	573.09	27.32	<0.001
	Group × time	131.87	2	65.94	3.14	0.054
	Error	9732.62	464	20.98		
	Between-group	820.63	1	820.63	5.41	0.021
Serum creatinine (mg/dL)	Within group	0.53	2	0.26	10.82	<0.001
	Group × time	0.05	2	0.03	1.04	0.356
	Error	11.27	464	0.02		
	Between-group	0.31	1	0.31	1.11	0.294

SS: sum of square; MS: Mean square; DF: degree of freedom; GFR: glomerular filtration rate. P<0.05 was significant

indicated that furosemide combination with standard hydration did not significantly lower the occurrence of CIN after coronary intervention.

It is shown that furosemide may decrease renal vascular resistance in the kidneys, inducing blood flow of the kidneys and generation of several metabolic alkalosis degrees that are shown to be correlated with a kidney preservative efficacy versus CIN.[25] Eventually, it prohibits the overload of the fluid, which shows superior clinical significance. Nevertheless, former clinical studies have highlighted that the net consequence of prophylactic furosemide appears to be an enhanced rate of CIN.[17]

In contrast to our findings, Gu *et al*.[9] reported that the administration of a low-dose amount of furosemide with sufficient hydration diminished CIN in candidates undergoing coronary angiography. They highlighted that preservative efficacies of furosemide were more obvious in a few populations including females and diabetic candidates, hypertensive subjects, those who take angiotensin-converting enzyme suppressor drug, PCI, impaired kidney in function, or those with high doses of contrast agent administration. Consistent with their findings, in our study, gender was not regarded as a risk factor between furosemide and control groups that represented CIN; However, in line with their results, in the present study, hypertension as well as diabetes mellitus were shown to be the most risk factors affecting CIN in both the groups. In line with our findings, Ghaffari *et al*. showed that hypercholesterolemia was one of the deterioration causes of CIN after PCI.[1] Cui *et al*. showed that furosemide administration after the assessment of efficient volume of blood can further decrease CIN occurrence represents the effectiveness of individualized precision management plans and is of note for further elevation in clinical practice.[26]

In contrast to positive outcomes regarding furosemide usage for preventing CIN, other researchers drew a deduction different from others. They imply that furosemide correlated with the enhanced CIN risk after radiologic contrast agent exposure.[16,17] Other investigations also showed the undesirable impact of furosemide.[18,27]

Results showed that BUN (mg/dL) was significantly higher in the furosemide group at 24 (P value = 0.048) and 72 h later (P value = 0.008). Based on the previous investigations, it can be caused by prerenal azotemia caused by diuretic effects.[28]

From broader clinical research, there is evidence suggesting that furosemide might work in preventing and treating acute renal failure. Yet, such discrepancy might vary depending on the patient population, sufficient or insufficient hydration, route of furosemide administration, and changes in the overall health of patients. Factors such as timing and dosage can also impact drug concentration, renal blood supply, renal medullary oxygen consumption, and the body's response to stress.

Study limitations

Limited budget in sample collection for the desired number of patients along with challenges in providing laboratory requirements were noted as study limitations of this study.

Conclusion

Using a low dose of furosemide along with standard hydration does not have an impact on lowering CIN in patients with STEMI undergoing coronary intervention. For a more precise assessment of this combination's effectiveness, future research should include randomized controlled clinical trials with sizable and high-quality participant samples.

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Consent for publication

All candidates provided their informed consent.

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Conflicts of interest

There are no conflicts of interest.

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