Contrast-Induced Nephropathy in Patients With ST-Segment Elevation Myocardial Infarction

Is it Affected by Treatment Strategy?

Mohamed Khalfallah, Mohamed Abdalaal, Mona Adel

Tanta, Egypt

ABSTRACT

Background: Contrast-induced nephropathy (CIN) is a frequent complication after percutaneous coronary intervention (PCI) and severely affects morbidity and mortality, especially in patients with ST-segment elevation myocardial infarction.

Objective: This study sought to determine the incidence, risk factors, and in-hospital outcome of CIN in patients with ST-segment elevation myocardial infarction managed by pharmacoinvasive strategy (PIS) versus those managed by primary PCI (PPCI).

Methods: The study was conducted on 670 patients with ST-segment elevation myocardial infarction divided into 2 groups: group I (PPCI group) and group II (PIS group), the 2 groups were compared with each other for the incidence of CIN, risk factors, and in-hospital major adverse cardiac events.

Results: The incidence of CIN in the PIS group (30 patients, 8.8%) was lower than PPCI group (36 patients, 10.9%); however, there was no statistically significant difference between the 2 groups ($p = 0.365$). Multivariate regression analysis showed that advanced age >60 years (odds ratio [OR] = 4.453; 95% confidence interval [CI]: 2.489 to −7.967; $p = 0.001$), history of diabetes mellitus (OR = 2.366; 95% CI: 1.298 to −4.315; $p = 0.005$) and hypertension (OR = 1.930; 95% CI: 1.053 to −3.539; $p = 0.034$), volume of contrast agent >180 ml (OR = 2.276; 95% CI: 1.290 to −4.016; $p = 0.005$), and cardiogenic shock (OR = 4.098; 95% CI: 1.726 to −9.728; $p = 0.001$) were the independent predictors of CIN. Mortality and major adverse cardiac events were significantly higher in patients with CIN.

Conclusions: The incidence of CIN was slightly lower in PIS as compared to PPCI; however, this reduction was not statistically significant. The independent predictors of CIN were advanced age, history of diabetes mellitus and hypertension, high dose of contrast agent, and cardiogenic shock.

Contrast-induced nephropathy (CIN) is a procedure-related renal injury that follows intravascular administration of radiopaque contrast media in susceptible individuals [1], also known as contrast-induced acute kidney injury (AKI). CIN remains responsible for large proportion of hospitalized patients with AKI [2,3], and its incidence varies between the general populations and increased up to 50% of high-risk subgroups following coronary angiography or percutaneous coronary intervention (PCI) [1]. It has been shown that CIN is associated with adverse outcome, increased mortality, cardiovascular events, renal failure, and prolonged hospitalization [4].

Primary PCI (PPCI) is the treatment of choice for patients with ST-segment elevation myocardial infarction (STEMI) according to the recent guidelines, and if a PCI-capable center is not immediately available, the alternative approach is pharmacoinvasive strategy (PIS), which involves fibrinolysis at the point of contact with a non-PCI-capable center, followed by transfer of the patient to a PCI-capable center within 3 to 24 h of fibrinolysis. Clinical trials and registry data have shown that clinical outcomes with PIS are comparable to those with PPCI [5–7]. The widespread embracing of PPCI has increased the incidence of CIN due to some difficulties in rapidly assessing CIN risk, usage of prophylactic measures and dealing with hemodynamic compromise that may lead to the occurrence of CIN [8]. The main pitfall in patients undergoing PPCI is that renal function is often unknown at the time of contrast media exposure because PPCI has to be performed without delay, leaving no time for renal function assessment. Moreover, the short delay between patient admission and PPCI significantly limits the use of renal

The authors report no relationships that could be construed as a conflict of interest.

From the Department of Cardiology, Tanta University Hospital, Tanta, Egypt. Correspondence: M. Khalfallah (khalfallah@yahoo.com).

GLOBAL HEART

VOL. 14, NO. 3, 2019
ISSN 2211-8160/$36.00.
https://doi.org/10.1016/j.gheart.2019.07.001

protective measures, such as intravenous hydration. We hypothesized that patients with STEMI undergoing PIS will have less incidence of CIN, as the time delay from the intake of fibrinolytic therapy until PCI will be used to assess the patients' risk to develop CIN, to start prophylactic hydration if high risk, and to recommend less contrast volumes during PCI. So the objective of the present study was to determine the incidence of CIN in patients with STEMI managed by PIS and to compare it with those managed by PPCI, also to determine risk factors for CIN, and to compare the incidence of in-hospital major adverse cardiac events (MACEs) between both groups.

PATIENTS AND METHODS

This is a prospective study of patients with STEMI, who underwent revascularization by either PPCI or PIS. The patients were admitted to our cardiovascular department for PPCI or transferred from other surrounding (non-PCI-capable) hospitals for PIS during the period from January 1, 2017, to December 31, 2018.The study was conducted on 670 patients with STEMI who were classified into 2 groups: group I patients were managed with PPCI (PPCI group); and group II patients were managed with PIS (PIS group). All patients gave written informed consent, and the study was approved by the local ethical committee.

Exclusion criteria

All patients with STEMI who were not eligible for revascularization as late presentation after 24 h, patients who were exposed to contrast media within the last 72 h, and patients on renal dialysis were excluded.

All patients were interrogated and clinically examined immediately after hospital admission with special emphasis on risk factors, comorbidities, and medication use. Body mass index and hemodynamic data (systolic and diastolic blood pressure and heart rate) were also collected. Patients were considered to be at high risk for CIN if they had chronic kidney disease with high baseline serum creatinine and if they were diabetic, hypertensive, anemic, or elderly [9].

Laboratory investigation

Complete blood count, random blood sugar, lipid profile, blood urea, and serum uric acid were measured. Baseline serum creatinine concentrations were measured from blood samples obtained immediately after hospital admission or were obtained from the transferred patients' files. Measurements were repeated at 24, 48, and 72 h, and creatinine clearance was calculated using the modified Modification of Diet in Renal Disease equation. CIN was defined as a relative ($\geq$25%) or absolute ($\geq$0.5 mg/dl) increase in serum creatinine from baseline within 3 days after contrast media exposure. Chronic renal failure was defined, according to the recommendations of the European Society of Nephrology, as estimated glomerular filtration rate (eGFR) <60 ml/min/1.73 m^2, measured with the modified Modification of Diet in Renal Disease formula [10].

Patients with STEMI were subjected to PPCI immediately after admission, and nonionic iso-osmolar contrast media was used for all patients. However, patients with STEMI subjected to PIS were assessed properly, and patients with risk factors for CIN or with high baseline serum creatinine received less contrast media and were hydrated with intravenous 0.9% saline during PCI at a rate of 1 ml/kg/h or 0.5 ml/kg/h in patients with cardiogenic shock, heart failure, or left ventricular ejection fraction <40%, and continued for 12 to 24 h after the procedure. The primary endpoint of the study was the occurrence of CIN and each group was subsequently subdivided into 2 subgroups according to the occurrence of CIN. The secondary endpoint was the occurrence of MACEs; the patients were followed up during hospital stay for cardiogenic shock, heart failure, major bleeding, cardiac arrest, and cerebral stroke.

Statistical analysis

Statistical analysis was done using SPSS version 23 (IBM, Armonk, New York). Quantitative data were expressed as mean $\pm$ SD. Categorical data were expressed as absolute values and percentage. Student's *t*-test was used to test significance between the 2 groups in quantitative data, and for categorical variables chi-square test of significance was used. A p value < 0.05 was considered statistically significant. Multivariate regression analysis using binary logistic regression was performed to detect the independent predictors of CIN.

RESULTS

The study was conducted on 670 patients who presented with STEMI. Patients were divided into 2 groups according to the method of management, group I (PPCI group) included 330 patients and group II (PIS group) included 340 patients. The main finding of the present study was that the incidence of CIN in the PIS group was lower than that in the PPCI group; as 30 patients (8.8%) developed CIN in the PIS group versus 36 patients (10.9%) in the PPCI group, but there was no statistically significant difference between the 2 groups ($p = 0.365$). There was a statistically significant difference between both groups regarding left ventricular ejection fraction, which was lower in group II than in group I ($p = 0.018$). There were no statistically significant differences between both groups regarding age, sex, diabetes mellitus, hypertension, smoking, dyslipidemia, peripheral vascular disease, and chronic kidney disease. The site of coronary artery lesion, volume of contrast media used during intervention, the eGFR before and after the procedure, and the occurrence of in-hospital MACEs showed no statistically significant differences between both groups as shown in Table 1.

Subgroup analysis for group I (PPCI group) showed that patients with CIN (group IB) were advanced in age and had more prevalence of diabetes mellitus, hypertension, chronic

TABLE 1. Demographics, clinical characteristics, laboratory results, and angiographic results of all patients in the 2 groups

	Group I (PPCI Group) (n = 330; 49.3%)	Group II (PIS Group) (n = 340; 50.7%)	p Value
Age, yrs	60.0 ± 8.17	59.3 ± 8.76	0.267
Male	184 (55.8)	165 (48.5)	0.061
Hypertension	110 (33.3)	131 (38.5)	0.161
Diabetes mellitus	130 (39.4)	121 (35.6)	0.309
Smoking	77 (23.3)	93 (27.4)	0.232
Dyslipidemia	114 (34.5)	107 (31.5)	0.397
PVD	69 (20.9)	70 (20.6)	0.918
CKD	47 (14.2)	55 (16.2)	0.486
BMI, kg/m^2	24.04 ± 4.18	24.63 ± 4.71	0.084
Systolic BP, mm Hg	105.2 ± 16.07	104.2 ± 16.18	0.462
Diastolic BP, mm Hg	65.8 ± 9.39	65.7 ± 9.39	0.896
Atrial fibrillation	42 (12.7)	48 (14.1)	0.598
LVEF, %	46.3 ± 4.71	45.4 ± 4.99	0.018*
Total cholesterol, mg/dl	206.4 ± 79.6	199.1 ± 77.9	0.288
HDL, mg/dl	38.7 ± 8.39	39.2 ± 8.48	0.430
LDL, mg/dl	131.8 ± 27.6	132.7 ± 27.3	0.678
Triglycerides, mg/dl	161.1 ± 36.2	161.4 ± 36.8	0.912
Non-HDL cholesterol, mg/dl	167.7 ± 81.9	159.9 ± 79.5	0.212
Serum uric acid, mg/dl	5.52 ± 1.45	5.53 ± 1.40	0.942
Hemoglobin, g/dl	11.61 ± 1.44	11.66 ± 1.41	0.633
Random blood sugar, mg/dl	198.6 ± 88.45	196.2 ± 81.66	0.720
Creatinine pre-procedure, mg/dl	1.04 ± 0.24	1.04 ± 0.25	0.802
Creatinine post-procedure, mg/dl	1.16 ± 0.42	1.15 ± 0.42	0.822
eGFR pre-procedure, ml/min/1.73 m^2			
≥60	242 (73.3)	244 (71.8)	0.649
30–59	88 (26.7)	96 (28.2)	
eGFR post-procedure, ml/min/1.73 m^2			
≥60	220 (66.7)	226 (66.5)	0.882
30–59	94 (28.5)	100 (29.4)	
<30	16 (4.8)	14 (4.1)	
eGFR pre-procedure	56.96 ± 6.44	56.21 ± 7.46	0.162
eGFR post-procedure	54.42 ± 10.32	54.25 ± 10.46	0.840
CIN	36 (10.9)	30 (8.8)	0.365
Volume of contrast agent, ml	183.3 ± 68.30	181.8 ± 64.15	0.773
LM coronary artery	7 (2.1)	6 (1.8)	0.738
LAD coronary artery	130 (39.4)	124 (36.5)	0.436
CX coronary artery	91 (27.6)	110 (32.4)	0.177
Right coronary artery	103 (31.2)	100 (29.4)	0.612
Mortality	20 (6.1)	22 (6.5)	0.827
Cardiogenic shock	24 (7.3)	25 (7.4)	0.968
Heart failure	35 (10.6)	40 (11.8)	0.634
Cardiac arrest	15 (4.5)	18 (5.3)	0.654
Major bleeding	8 (2.4)	11 (3.2)	0.527
Cerebral stroke	3 (0.9)	2 (0.6)	0.629

Values are mean ± SD or n (%).

BMI, body mass index; BP, blood pressure; CIN, contrast-induced nephropathy; CKD, chronic kidney diseases; CX, circumflex; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LAD, left anterior descending; LDL, low-density lipoprotein; LM, left main; LVEF, left ventricular ejection fraction; PIS, pharmacoinvasive strategy; PPCI, primary percutaneous coronary intervention; PVD, peripheral vascular diseases.

*Statistically significant.

TABLE 2. Demographics, clinical characteristics, laboratory results, and angiographic results of all patients in PPCI subgroups

	Group IA (No AKI) (n = 294; 89.1%)	Group IB (AKI) (n = 36; 10.9%)	p Value
Age, yrs	59.5 ± 8.04	63.9 ± 8.35	0.003*
Male	164 (55.8)	20 (55.6)	0.979
Hypertension	91 (31.0)	19 (52.8)	0.009*
Diabetes mellitus	107 (36.4)	23 (63.9)	0.001*
Smoking	67 (22.8)	10 (27.8)	0.504
Dyslipidemia	101 (34.4)	13 (36.1)	0.834
PVD	56 (19.0)	13 (36.1)	0.017*
CKD	35 (11.9)	12 (33.3)	0.001*
Systolic BP, mm Hg	105.7 ± 15.35	100.7 ± 20.66	0.068
Diastolic BP, mm Hg	66.1 ± 8.82	63.5 ± 15.16	0.129
LVEF, %	46.3 ± 4.64	45.7 ± 5.28	0.475
Hemoglobin, g/dl	11.6 ± 1.46	11.4 ± 1.29	0.275
Random blood sugar, mg/dl	199.1 ± 90.56	193.6 ± 69.70	0.723
Creatinine pre-procedure, mg/dl	1.02 ± 0.23	1.15 ± 0.29	0.003*
Creatinine post-procedure, mg/dl	1.05 ± 0.23	2.01 ± 0.61	0.001*
eGFR pre-procedure, ml/min/1.73 m^2			
≥ 60	222 (75.5)	20 (55.6)	0.011*
30–59	72 (24.5)	16 (44.4)	
E-GFR post-procedure, ml/min/1.73 m^2			
≥ 60	220 (74.8)	0 (0.0)	0.001*
30–59	74 (25.2)	20 (55.6)	
<30	0 (0.0)	16 (44.4)	
eGFR pre-procedure	57.35 ± 6.03	53.78 ± 8.61	0.002*
eGFR post-procedure	57.17 ± 6.24	31.94 ± 9.59	0.000*
Volume of contrast agent, ml	179.9 ± 65.82	210.6 ± 82.04	0.011*
LM coronary artery	5 (1.7)	2 (5.6)	0.130
LAD coronary artery	117 (39.8)	13 (36.1)	0.669
CX coronary artery	82 (27.9)	9 (25.0)	0.714
Right coronary artery	91 (31.0)	12 (33.3)	0.771
Mortality	15 (5.1)	5 (13.9)	0.037*
Cardiogenic shock	18 (6.1)	6 (16.7)	0.021*
Heart failure	25 (8.5)	10 (27.8)	0.001*
Cardiac arrest	11 (3.7)	4 (11.1)	0.045*
Major bleeding	7 (2.4)	1 (2.8)	0.884
Cerebral stroke	2 (0.7)	1 (2.8)	0.211

Values are mean ± SD or n (%).
AKI, acute kidney injury; other abbreviations as in Table 1.
*Statistically significant.

kidney disease, and peripheral vascular disease than patients without CIN (group IA) as shown in Table 2. They also had lower eGFR than those of group IA, whereas creatinine pre and post-PCI was higher in group IB than in patients in group IA. The volume of contrast media was higher in group IB. As regards MACEs, mortality, cardiogenic shock, heart failure, and cardiac arrest occurred more frequently in group IB, and there were statistically significant differences between both subgroups (p = 0.037, 0.021, 0.001, and 0.045, respectively) as shown in Table 2. Subgroup analysis for group II (PIS group) showed that patients with CIN (group IIB) were older, and they had lower eGFR and systolic blood pressure than those without CIN (group IIA). Creatinine pre- and post-PCI was significantly higher in group IIB. As regards MACEs, cardiogenic shock, heart failure, and cerebral stroke occurred more frequently in group IIB, and there were statistically significant differences between both subgroups (p = 0.039, 0.001, and 0.039, respectively) as shown in Table 3.

Univariate and multivariate regression analysis were performed to detect the independent predictors of CIN and showed that advanced age >60 years (odds ratio [OR] = 4.453; 95% confidence interval [CI]: 2.489 to 7.967; p = 0.001), presence of diabetes mellitus (OR = 2.366; 95% CI: 1.298 to 4.315; p = 0.0005), and hypertension

TABLE 3. Demographics, clinical characteristics, laboratory results, and angiographic results of all patients in PIS subgroups

	Group IIA (No AKI) (n = 310; 91.2%)	Group IIB (AKI) (n = 30; 8.8%)	p Value
Age, yrs	58.9 ± 8.68	63.7 ± 8.44	0.004*
Male	149 (48.1)	16 (53.3)	0.581
Hypertension	117 (37.7)	14 (46.7)	0.338
Diabetes mellitus	104 (33.5)	17 (56.7)	0.012*
Smoking	84 (27.1)	9 (30.0)	0.733
Dyslipidemia	94 (30.3)	13 (43.3)	0.143
PVD	63 (20.3)	7 (23.3)	0.697
CKD	48 (15.5)	7 (23.3)	0.265
Systolic BP, mm Hg	104.8 ± 16.17	98.3 ± 15.33	0.036*
Diastolic BP, mm Hg	65.8 ± 9.46	64.8 ± 8.66	0.599
LVEF, %	45.4 ± 4.94	44.8 ± 5.61	0.549
Hemoglobin, g/dl	11.67 ± 1.41	11.60 ± 1.38	0.802
Random blood sugar, mg/dl	195.6 ± 81.36	202.3 ± 85.81	0.667
Creatinine pre-procedure, mg/dl	1.03 ± 0.24	1.19 ± 0.27	0.001*
Creatinine post-procedure, mg/dl	1.06 ± 0.24	2.09 ± 0.67	0.001*
eGFR pre-procedure, ml/min/1.73 m^2			
≥60	228 (73.5)	16 (53.3)	0.019*
30–59	82 (26.5)	14 (46.7)	
eGFR post-procedure, ml/min/1.73 m^2			
≥60	226 (72.9)	0 (0.0)	0.001*
30–59	84 (27.1)	16 (53.3)	
<30	0 (0.0)	14 (46.7)	
eGFR pre-procedure	56.66 ± 6.92	51.50 ± 10.72	0.001*
eGFR post-procedure (M±SD) (mL/min/1.73m^2)	56.48 ± 7.11	31.27 ± 11.91	0.001*
Volume of contrast agent, ml	180.5 ± 63.27	195.7 ± 72.33	0.216
LM coronary artery	6 (1.9)	0 (0.0)	0.442
LAD coronary artery	116 (37.4)	8 (26.7)	0.243
CX coronary artery	98 (31.6)	12 (40.0)	0.348
Right coronary artery	90 (29.0)	10 (33.3)	0.622
Mortality	20 (6.5)	2 (6.7)	0.964
Cardiogenic shock	18 (5.8)	7 (23.3)	0.001*
Heart failure	33 (10.6)	7 (23.3)	0.039*
Cardiac arrest	17 (5.5)	1 (3.3)	0.615
Major bleeding	10 (3.2)	1 (3.3)	0.975
Cerebral stroke	1 (0.3)	1 (3.3)	0.039*

Values are mean ± SD or n (%).
Abbreviations as in Tables 1 and 2.
*Statistically significant.

(OR = 0.1930; 95% CI: 1.053 to −3.539; p = 0.034). Volume of contrast agent >180 ml (OR = 0.2276; 95% CI: 1.290 to 4.016; p = 0.005) and cardiogenic shock (OR = 4.098; 95% CI: 1.726 to −9.728; p = 0.0001) were the independent predictors of CIN as shown in Table 4 and Figure 1.

DISCUSSION

We hypothesized that CIN will be less prevalent in patients going for PIS as compared to PPCI, so in this study's 2-year duration, we prospectively studied 330 patients going for PPCI (group I) and compared them with 340 patients going for PIS (group II). For the first time, the current study showed that patients with STEMI going for PIS have less incidence for CIN as compared to those going for PPCI (8.8% vs. 10.9%); this maybe attributed to the fact that when patients with PIS arrived to the hospital, there was no need to rush them for coronary angiography. So they were properly evaluated and high-risk patients for CIN were identified. The high-risk patients for CIN were then properly managed by prophylactic intravenous hydration along with advice to the team to use the least amount of contrast media as much as possible, and hemodynamic stability was achieved before PCI. In spite of these

TABLE 4. Univariate and multivariate regression analysis showing the independent predictors of CIN

	Univariate Analysis			Multivariate Analysis		
	OR	(95% CI)	p Value	OR	(95% CI)	p Value
Age >60 yrs	4.742	(2.747–8.184)	0.001*	4.453	(2.489–7.967)	0.001*
Diabetes mellitus	2.865	(1.701–4.826)	0.001*	2.366	(1.298–4.315)	0.005*
Hypertension	1.904	(1.142–3.173)	0.014*	1.930	(1.053–3.539)	0.034*
PVD	1.772	(1.010–3.108)	0.046*	1.093	(0.494–2.416)	0.827
CKD	2.538	(1.419–4.537)	0.002*	1.854	(0.814–4.222)	0.142
Volume of contrast agent >180 ml	1.961	(1.176–3.269)	0.010*	2.276	(1.290–4.016)	0.005*
Cardiogenic shock	3.870	(1.934–7.746)	0.001*	4.098	(1.726–9.728)	0.001*
Heart failure	3.266	(1.767–6.038)	0.001*	2.025	(0.955–4.292)	0.066
Cardiac arrest	1.686	(0.628–4.527)	0.300	1.116	(0.348–3.577)	0.853

CI, confidence interval; OR, odds ratio; other abbreviations as in Table 1.
*Statistically significant.

procedures, the difference between the 2 groups did not reach statistical significance (p = 0.365) because the patients received intravenous hydration ongoing to the catheterization laboratory or immediately before, but intravenous hydration is recommended at least 12 h before the procedure [11]. Moreover, presence of hypotension, hemodynamic instability, or cardiogenic shock are additional contributing factors that may lead directly to AKI.

The incidence of CIN varies greatly after coronary angiography and may be as low as 6% in patients undergoing elective catheterization [12]. The incidence of CIN in the study of Tziakas et al. [13] was about 16% in both elective and urgent catheterization, and it reached 25% in urgent catheterization [14]. Mehran et al. [15] showed that the overall occurrence of CIN in 8,357 patients was 13.1%. Bouzas-Mosquera et al. [16] and Gohbara et al. [17] reported that the incidence of CIN was 12% in a high-risk group of patients undergoing PPCI.

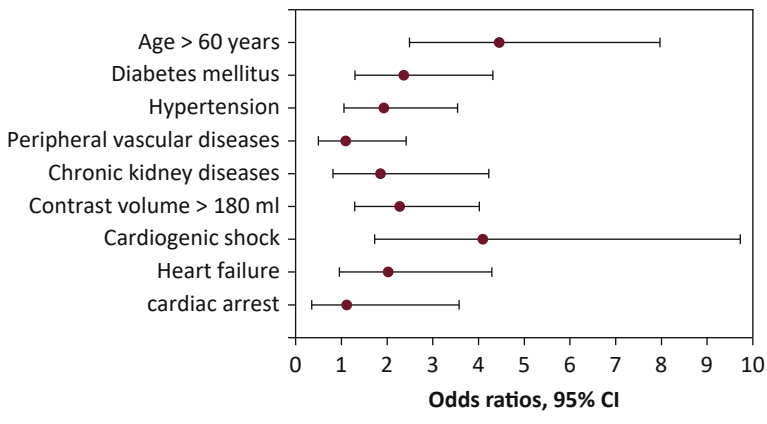

FIGURE 1. Forest plot of the multivariate regression analysis showing odds ratios and 95% confidence interval (CI) of the independent predictors of contrast-induced nephropathy.

There are multiple risk factors for CIN after PCI, and identification of patients at high risk for CIN occurrence plays an important role in the outcome and prognosis. Several previous studies [18–24] had reported predictors of CIN including advanced age, elevated baseline serum creatinine, heart failure, reduced ejection fraction, anemia, high-sensitivity C-reactive protein, hyperglycemia, and high doses of contrast media. In the current study, the independent predictors of CIN were advanced age 60 years, history of diabetes mellitus and hypertension, volume of contrast agent >180 ml, and cardiogenic shock. Similarly, in clinical studies including elective procedures, the presence of CIN largely depends on coexisting risk factors such as intravascular volume depletion, baseline renal dysfunction, heart failure, and diabetes mellitus [25].

Contrast-induced nephropathy implies impairment of renal function occurring within 3 to 5 days following the administration of contrast media in the absence of an alternative etiology [26]. However, it is unlikely that contrast media is the sole factor responsible for AKI in the patients undergoing PCI. Conditions resulting in hypotension, hemodynamic instability, or cardiogenic shock and acute heart failure are additional contributing factors that may lead directly to AKI or potentiate the effects of contrast media [14]. Consequently, in the setting of PCI, most AKI events likely have alternative and multiple etiologies. However, there is association between high dose of contrast media and increased incidence of CIN in patients undergoing PCI. This is strengthened by the results of our study in which patients with CIN received higher doses of contrast media and the p value was statistically significant in PPCI group (p = 0.011). Also our result was comparable with that of Kooiman et al. [27], who reported that high dose of contrast was a marginal predictor of CIN after PCI, with an estimated attributable risk fraction to CIN of 10.6%. Several studies also reported a positive correlation between higher dose of contrast media and increased incidence of CIN in patients undergoing PCI [28,29]. However, Gohbara et al. [17] did not find a relation

between the volume of contrast agents and CIN onset and this may be attributed to the fewer patients included in their study.

In the current study, patients with CIN had a higher mortality (group IB, 13.9% and group IIB, 6.7%) than did patients without CIN (group IA, 5.1% and group IIA, 6.5%). Moreover, the incidence of heart failure was significantly higher in patients with CIN (group IB, 27.8% and group IIB, 23.3%) versus (group IA, 8.5% and group IIA, 10.6%), also the incidence of cardiogenic shock was significantly higher in patients with CIN. Our results were comparable with previous studies demonstrated mortality and outcome in patients with STEMI undergoing either PPCI or PIS [28—31]. Bouzas-Mosquera et al. [16] reported 14% mortality in patients with CIN after PPCI. Gohbara et al. [17] reported in-hospital mortality of 11.4%.

Our study recommended the following: 1) All patients with acute STEMI going for PPCI or PIS should be screened for risk factors of CIN. 2) Patients with multiple risk factors should be considered as high-risk candidates to develop CIN. For high-risk candidates, 3) preventive measures should be started immediately after admission with injection of the lowest possible volume of contrast agents and 4) complex procedures should be avoided.

Study limitations

Our study is an observational study, and the patients were enrolled at a single center. Number of the patients in this study was relatively small, and we need large numbers in different centers to represent the whole population. Patients admitted to our center directly and subjected to PPCI may have better outcome regarding MACEs than did patients who were admitted to surrounding non-PCI-capable hospitals and received fibrinolytic therapy before they were transferred to us for PIS. Long-term mortality and MACEs were not detected as we followed the patients only during hospital stay.

Future directions

In spite of these limitations, this study opened the door for further multicenter randomized controlled trials to validate the results with a large proportion of the population.

CONCLUSIONS

The incidence of CIN was reduced in patients with STEMI revascularized by PIS as compared to PPCI; however, this reduction was not statistically significant. Risk factors for CIN were advanced age >60 years, history of diabetes mellitus or hypertension, and cardiogenic shock. Use of large amount of contrast media during the procedure was also incriminated. Patients with CIN had higher rates of in-hospital mortality and MACEs.

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