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ORIGINAL PAPER

Contrast medium induced nephropathy in patients undergoing percutaneous coronary intervention for acute coronary syndrome: differences in STEMI and NSTEMI

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Abstract The aim of this study was to assess the incidence, clinical predictors, and outcome of patients developing contrast medium induced nephropathy (CIN) after percutaneous coronary intervention (PCI) for acute coronary syndrome (ACS).

Background CIN is associated with significant higher morbidity and mortality after coronary intervention. Recently it was shown, that patients undergoing percutaneous coronary intervention for acute myocardial infarction have a significant higher risk of developing CIN. Non-ST-elevating myocardial infarction (NSTEMI) patients (pts) might be at an even higher risk developing CIN than patients with ST-elevating myocardial infarction (STEMI), because of presenting older and more often with diabetes.

Methods In 392 consecutive ACS patients developing myocardial infarction and therefore undergoing emergent coronary angiography between October 2004 and March 2007, we measured serum creatinine concentration (Cr) at baseline and each day of the following 3 days. Contrast medium induced nephropathy was defined as an increase in Cr > 0.5 mg/dl. ACS was defined according to the guidelines of the German Society of Cardiology.

Results Overall, 392 pts were included: 203 (51.8%) with STEMI and 189 (48.2%) with NSTEMI. Patients with STEMI developed more often a cardiogenic shock (18 vs. 6%; $P < 0.001$) whereas patients with NSTEMI were older (67 vs. 61 years; $P < 0.001$) and presenting with a higher co-morbidity. Forty-five (11.5%) pts developed CIN; 22 (10.8%) in the STEMI group and 23(12.2%) in the NSTEMI group ($P = 0.75$). Patients developing CIN presented a more complicated clinical course and a significantly longer hospital stay (14 vs. 10 days; $P < 0.001$). The mortality rate was also significantly higher (16 vs. 6%; $P < 0.05$).

Conclusion This prospective study showed no differences in the incidence of developing CIN in patients undergoing PCI for STEMI or NSTEMI, but the predisposing factors, however, differed significantly. Although STEMI patients needed significantly more contrast medium for revascularisation, they did not develop CIN more often. CIN was associated with higher in-hospital complication rate and mortality. Thus, better preventive strategies according to the different predisposing factors leading to CIN are needed to reduce morbidity and mortality, especially in high risk patients.

Keywords Contrast induced nephropathy · Acute coronary syndrome · Percutaneous coronary intervention · NSTEMI · STEMI

Abbreviations

ACS	Acute coronary syndrome
AMI	Acute myocardial infarction

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CAD Coronary artery disease
CCU Coronary care unit
CIN Contrast induced nephropathy
Cr Creatinine clearance
PCI Percutaneous coronary intervention

Introduction

Contrast medium induced nephropathy (CIN) is a possible complication of invasive coronary diagnostic and intervention. Recently it was shown that patients with acute myocardial infarction undergoing percutaneous coronary intervention (PCI) shows a significantly increased risk developing CIN in comparison to patients undergoing elective contrast medium exposure [18]. The development of CIN has been associated with increased in-hospital and long term morbidity and mortality, prolonged hospitalization, and long term renal impairment [3, 6, 10, 19, 34]. A lot of risk factors have been identified. Chronic renal insufficiency, diabetes mellitus, congestive heart failure, intravascular volume depletion, and the use of a large contrast medium amount are considered to be important predisposing factors [22, 27].

However, patients treated with PCI for ACS are at higher risk for CIN. Several conditions may contribute to renal injury in this setting. Among them, hypotension or even cardiogenic shock, a large volume of contrast medium and the lack of starting a state of the art renal prophylactic therapy in case of pre-existing renal failure are the factors most likely involved. Recently published studies demonstrated that renal insufficiency and acute myocardial infarction (AMI) are a high risk combination for the development of acute renal failure [28, 30, 33, 36]. Some studies even showed that renal dysfunction is an independent risk factor for death in AMI [10–12].

Moreover, NSTEMI patients in contrast to patients with STEMI are older and have an extended cardiac co-morbidity and extra cardiac diseases like pre-existing renal failure and diabetes mellitus as well as a higher atherosclerotic burden. This may suggest that these patients are at even higher risk developing CIN after percutaneous intervention than patients with STEMI [4, 21].

The purpose of this prospective study was to determine the incidence, the clinical predictors, and the clinical consequences of CIN in patients undergoing PCI for ACS. Furthermore, differences in the clinical course, complications, and outcome in patients with STEMI and NSTEMI developing CIN are discussed.

Methods

Study population

Between October 1, 2004 and March 30, 2007 we enrolled all consecutive patients admitted to the coronary care unit (CCU) for ACS who were treated with emergent coronary intervention. According to the German guideline [12] patients were included to this study with characteristic chest pain lasting for at least 30 min with electrocardiographic ST-segment elevation (STEMI) of at least 0.2 mV in two or more contiguous leads, or left bundle branch block as well as patients with a positive Troponin I test but without ST-Segment elevation (NSTEMI). Patients were excluded if the coronary anatomy was not suitable for coronary intervention or if emergency bypass grafting was required.

Study protocol

Physiologic saline (0.9%) was given intravenously at a rate of 1 ml/kg/h for 12 h after contrast medium exposure. In patients with left ventricular dysfunction (ejection fraction <40%) or overt heart failure (Killip Class III and IV) the hydration rate was reduced to 0.5 ml/kg/h. The use of beta-adrenergic blocking agents, angiotension-converting enzyme inhibitors, platelet glycoprotein IIb/IIIa receptor inhibitors (abciximab, tirofiban, and eptifibatid), diuretics or the indication to intra-aortic balloon pump (IABP) or inotropic drug support was left to the direction of the interventional or ICU cardiologists according to our institute's clinical protocols based on international recommendations [32]. We performed an echocardiographic evaluation of the left ventricular ejection fraction in all subjects within 12 h from hospital admission. Serum creatinine concentration (Cr) was measured at the time of admission, before coronary intervention and every day for the following 3 days and at discharge from the ICU. Creatinine clearance was calculated by applying the Cockcroft formula to the serum creatinine concentration (Cr) [5]. During hospitalization the following adverse clinical events were considered: cardiogenic shock, acute renal failure, and death.

Coronary intervention

Coronary intervention was performed by a 24-h on-call emergency team, according to standard clinical practice, using the femoral approach and 6-F guiding catheters [2]. The low-osmolality nonionic contrast medium Iomeprol (Iomeron, Bracco s.p.a., Milan, Italy) with 350 mg/ml of iodine content was used in all cases. Patients received a

bolus of 5.000 U heparin, 500 mg of acetylsalicylic acid, and 300 mg of clopidogrel immediately before the procedure [9, 14, 37]. Contrast medium dose, angioplasty technique, and supportive pharmacologic therapies as well as ACT-measurements were left to the discretion of the interventional cardiologist.

Definitions

Contrast medium induced nephropathy was defined as an absolute increase in Cr > 0.5 mg/dl after coronary intervention [31]. Time to reperfusion was measured as the time from symptom onset to coronary reperfusion obtained with balloon inflation. Cardiogenic shock, complicating myocardial infarction was defined as prolonged hypotension (systolic blood pressure <85 mmHg) with evidence of decreased organ perfusion caused by severe left ventricular dysfunction, right ventricular infarction, or mechanical complications of infarction [7].

Statistical analysis

Continuous data are reported as the mean value ± SD unless otherwise specified. Categorical data are presented as absolute values and percentages. Comparison of continuous variables was performed by means of Students *t*-test. Chi square and fisher exact test were used for comparison of categorical variables as appropriate. A multivariate regression model was applied including all the potential confounding variables leading to CIN and death.

A P value <0.05 was considered to be statistically significant. All statistical analysis were performed with the Statistical Package for the Social Science version 14.0 (SPSS Inc., Chicago, Illinois).

Results

Incidence of CIN and clinical characteristics

Of a total of 456 ACS pts, 64 pts were excluded (22 because no intervention was necessary and 42 were treated with coronary bypass surgery). Hence, a total of 392 pts (280 men, 112 women; mean age 64 ± 13) were included in this study. Of them, 45 (11.5%) developed CIN. Table 1 shows the baseline clinical and procedural characteristics of patients with and without developing CIN. Patients with CIN were significantly older and more frequent of female gender, had a higher baseline Cr value, a longer time to reperfusion, and a lower left ventricular ejection fraction. In addition, they received a higher amount of contrast medium during PCI than patients without CIN. When creatinine clearance was calculated [5], 64 (13%) of the 392 pts had a moderately pre-existing impaired renal function (<60 ml/min). Of them 23 (36%) developed CIN after coronary intervention. The relationship between the development of CIN and preexisting renal failure is shown in Fig. 1.

The relation of CIN with in-hospital outcome

Patients developing CIN had a more complicated in-hospital clinical course. The average stay in hospital of patients who developed CIN was 1.5 times longer than in patients without CIN (15 ± 11 vs. 9 ± 6 days; $P < 0.001$). Furthermore they developed more frequently a cardiogenic shock (24 vs. 10%). The overall in hospital mortality rate in the entire population was 7.1%. The mortality rate was significantly higher (15.6 vs. 6.1%) in patients developing CIN.

Differences in STEMI and NSTEMI

A total of 392 pts presented with ACS at hospital admission: 203 (51.7%) pts had STEMI and 189 (48.3%) had NSTEMI. As shown in Table 2, for 162 (79.8%) pts of the STEMI group the index event was the first manifestation of coronary artery disease (CAD), whereas almost 40% of the pts in the other group had a history of CAD. The incidence of a three vessel disease was also significantly higher in the NSTEMI group (19.7 vs. 37%; $P < 0.001$). The mean age of the population group was 64 ± 13 years. Patients with NSTEMI tended to be older than patients with STEMI (67 vs. 61 years; $P < 0.001$). The amount of patients older than 75 years was also significantly higher in patients with NSTEMI (32.8 vs. 21.7%; $P < 0.05$). Patients with NSTEMI had more often diabetes mellitus (30.7 vs. 16.7%; $P < 0.001$). Moreover, the number of patients with pre-existing renal insufficiency was higher in the NSTEMI group, but not significantly (19 vs. 13.8%; $P = 0.102$).

However, patients with STEMI developed more often a cardiogenic shock (17.7 vs. 6.3%; $P < 0.001$) requiring the IABP (8.4 vs. 1.1%; $P < 0.001$). According to this, the highest total creatinkinasis as an index of the magnitude of myocardial infarction was also significantly higher in patients with STEMI (1,882 vs. 803 U/l; $P < 0.001$). In addition people with STEMI needed significantly more contrast medium during the intervention (253 vs. 215 ml; $P < 0.001$). None the less there was no significant difference in the number of patients developing CIN in the two groups (10.8 vs. 12.2%; $P = 0.75$).

Differences in STEMI and NSTEMI in patients developing CIN

As shown in Table 3, patients with NSTEMI developing CIN were much older (76 ± 10 vs. 68 ± 12; $P < 0.05$)

Table 1 Baseline clinical and procedural characteristics of the study patients

Characteristic	CIN ($n = 45$)	No-CIN ($n = 347$)	All patients ($n = 392$)	P value
Demographics				
Men (%)	27 (60)	253 (72.9)	280 (71.9)	0.80
Mean age, years (SD)	72 ± 12	63 ± 13	64 ± 13	<0.0001
Age > 75 years (%)	23 (51.1)	83 (23.9)	106 (27)	<0.0001
Cardiac risk factors				
Diabetes mellitus (%)	11 (24.4)	85 (24.5)	96 (24.5)	0.76
Current smoker (%)	17 (37.8)	143 (41.2)	160 (40.8)	0.74
Hypertension (%)	41 (91.1)	283 (81.6)	324 (82.7)	0.14
Dyslipidemia (%)	35 (77.8)	269 (77.5)	304 (77.6)	1.0
Adipositas (%)				
Laboratory parameters				
Serum Creatinine (mg/dl)	1.26 ± 0.62	0.91 ± 0.26	0.95 ± 0.34	<0.001
Serum Creatinine > 1.5 mg/dl	10 (22.2)	23 (6.6)	33 (8.4)	<0.001
Creatinine clearance (ml/h)	67 ± 47	99 ± 38	95 ± 41	<0.001
Creatinine clearance < 60 ml	23 (51.1)	41 (11.8)	64 (13)	<0.0001
Highest total creatine kinase (U/l)	1,387 ± 1,526	1,355 ± 1,955	1,359 ± 1,909	0.91
Hb (g/dl)	12.9 ± 2.1	14.6 ± 4.9	14.4 ± 1.7	<0.001
Cardiovascular parameters				
STEMI (%)	22 (48.9)	181 (52.2)	203 (51.8)	0.75
NSTEMI (%)	23 (51.1)	166 (47.8)	189 (48.2)	0.75
First cardiac event (%)	25 (55.6)	251 (72.3)	276 (70.4)	0.24
One vessel disease (%)	8 (17.8)	132 (38)	140 (35.7)	<0.005
Two vessel disease (%)	16 (35.6)	88 (25.4)	104 (26.5)	0.10
Three vessel disease (%)	16 (35.6)	94 (27.1)	110 (28.1)	0.15
Previous CABG (%)	5 (11.1)	33 (9.5)	38 (9.7)	0.48
Mean LVEF (%)	49.5 ± 9.3	57.1 ± 12.2	56 ± 12	<0.001
LVEF < 40%	11 (24.4)	40 (11.5)	51 (13)	<0.05
Contrast medium				
Contrast volume, ml (SD)	263 ± 119	231 ± 96	234.9 ± 99	<0.05
Contrast volume > 300 ml (%)	16 (35.6)	51 (14.7)	67 (17.1)	<0.005
Clinical course				
Hospital stay, days (SD)	14.56 ± 10.7	9.40 ± 5.5	9.9 ± 6.5	<0.001
Time-to-reperfusion (h)	6.96 ± 2.52	4.94 ± 2.99	5.17 ± 3.0	<0.001
Cardiogenic Shock (%)	11 (24.4)	37 (10.7)	48 (12.2)	<0.05
IABP (%)	3 (6.7)	16 (4.6)	19 (4.8)	0.46
Death (%)	7 (15.6)	21 (6.1)	28 (7.1)	<0.05

and had more often an impaired renal function before hospital admission (35 vs. 14%; $P = 0.16$). Furthermore most of the patients in the NSTEMI group had a preexisting coronary artery disease (56 vs. 22%) and a significant longer hospital stay (17 ± 12.6 vs. 11.9 ± 7.4 days; $P < 0.001$).

Patients with STEMI who developed CIN, showed more often a complicated clinical course including the development of a cardiogenic shock (31.8 vs. 17.4%) requiring the implantation of the IABP (13.6 vs. 0.0%; $P = 0.109$). Furthermore contrast medium volume was higher in STEMI (295 ± 120 ml) compared with NSTEMI (231 ± 112 ml). The overall in-hospital mortality of the entire population was 7.1% ($n = 28$). However, the mortality rate was significantly higher in patients with STEMI developing CIN compared to patients with NSTEMI with CIN (22.8 vs. 8.7%; $P < 0.05$).

Independent correlates of CIN

In multivariate analysis, the following variables remained significantly independent correlates of CIN: Age >75 years (odds ratio [OR] 0.39, 95% confidence interval [CI] 0.26–

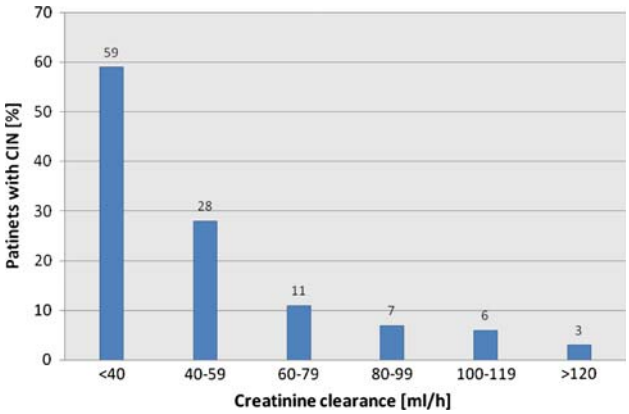

Fig. 1 Relationship between GFR and the development of CIN

1.10; $P = 0.001$), serum creatinine >1.5 mg/l (OR 4.33, CI 95% 1.34–13.95; $P = 0.014$), mean LVEF <40% (OR 2.07, CI 95% 0.85–5.05; $P = 0.001$), contrast volume >300 ml (OR 3.36, CI 95% 1.47–7.67; $P = 0.004$).

Independent correlates of death

In multivariate analysis, the following variables remained significantly independent correlates of death: CIN (OR 7.03, CI 95% 2.49–13.42; $P = 0.01$), cardiogenic shock (OR 3.83, CI 95% 1.10–13.42; $P = 0.035$) and highest total creatinkinasis (OR 1.37, CI 95% 1.08–1.73; $P = 0.008$).

Discussion

This study demonstrates that CIN is a frequent complication after invasive treatment of ACS, even in pts with normal baseline renal function. It is associated with increased in-hospital mortality and a prolonged hospitalization. Since several years acute PCI is the standard approach of acute STEMI. A lot of studies demonstrated, that survival and left ventricular function is preserved due to this procedure [15, 23, 24, 36]. The aim of this reperfusion strategy is to reduce mortality and morbidity. As a matter of principle this goal is best achieved by opening the infarct related artery as soon as possible [11, 12, 16, 20]. It is more difficult to diagnose NSTEMI in comparison with STEMI due to the time delay verifying various laboratory parameters. In future this time could be used for strategies preventing CIN, like hydration and the application of acetylcystein or sodium bicarbonate for example [25, 26, 29].

In our study population, the in-hospital mortality rate was 7.1%, a value comparable with those reported in other clinical trials [15]. In this prospective analysis, we have

Table 2 Differences in STEMI and NSTEMI

Characteristic	STEMI ($n = 203$)	NSTEMI ($n = 189$)	P value
Demographics			
Men (%)	137 (67.5)	143 (75.7)	0.46
Age, years (SD)	61 ± 13	67 ± 12	<0.001
Age > 75 years	44 (21.7)	62 (32.8)	<0.05
Cardiac risk factors			
Hypertension (%)	163 (80.3)	161 (85.2)	0.230
Current smoker (%)	98 (48.3)	62 (32.8)	<0.05
Diabetes (%)	34 (16.7)	58 (30.7)	<0.001
Dyslipidaemia (%)	147 (72.4)	157 (83.1)	<0.05
Laboratory parameters			
Creatinine clearance < 60 ml	28 (13.8)	36 (19)	0.102
Highest total creatine kinase (U/L)	1,882 ± 1,941	803 ± 1,711	<0.001
Clinical course			
Contrast volume, ml (SD)	253 ± 100	215 ± 94	<0.001
Cardiogenic shock (%)	36 (17.7)	12 (6.3)	<0.001
IABP (%)	17 (8.4)	2 (1.1)	<0.001
Death (%)	22 (10.8)	6 (3.2)	<0.05
CIN (%)	22 (10.8)	23 (12.2)	0.75
Cardiovascular parameters			
First cardiac event (%)	162 (79.8)	114 (60.3)	<0.001
Three vessel disease (%)	40 (19.7)	70 (37)	<0.001
Previous CABG (%)	8 (3.9)	30 (15.9)	<0.001
Mean LVEF (%)	57 ± 12	56 ± 13	0.33

Table 3 Differences in STEMI and NSTEMI in patients developing CIN

Characteristic	STEMI ($n = 22$)	NSTEMI ($n = 23$)	All patients ($n = 45$)	P value
Women (%)	8 (36.4)	10 (43.5)	18 (40)	0.763
Mean age, years (SD)	68.7 ± 12.4	75.7 ± 9.7	72.2 ± 11.5	<0.05
Age > 75 years (%)	7 (31.8)	16 (69.9)	22 (48.9)	<0.05
Serum creatinine > 1.5 mg/dl	3 (13.6)	8 (34.8)	11 (24.4)	0.16
Creatinine Clearance < 60 ml (%)	9 (40.9)	14 (60.9)	23 (51.1)	0.23
First cardiac event (%)	15 (68.2)	10 (43.5)	25 (55.6)	0.085
Three vessel disease (%)	6 (27.3)	10 (43.5)	16 (35.6)	0.20
LVEF < 40%	5 (22.7)	6 (26.1)	11 (24.4)	1.00
Cardiogenic shock (%)	7 (31.8)	4 (17.4)	11 (24.4)	0.31
IABP (%)	3 (13.6)	0 (0)	3 (13.6)	0.109
Death (%)	5 (22.7)	2 (8.7)	7 (156)	0.243
Hospital stay, days SD	11.9 ± 7.4	17 ± 12.6	14.6 ± 10.7	0.11

focused our data evaluation on the development of CIN during ACS as a possible complication of primary coronary intervention. Furthermore we pointed out the predisposing factors for developing CIN in STEMI and NSTEMI. Acute renal deterioration occurred in 11.5% ($n = 45$) of all patients undergoing coronary intervention and it was a strong predictor of in hospital morbidity and mortality. Patients with CIN had a more prolonged and complicated clinical course and a significant higher in-hospital mortality (15.6 vs. 6.1%).

In patients with ACS undergoing an emergent coronary intervention there are two well-known reasons for developing acute renal impairment. The first one is the direct toxicity of contrast medium [13] and the second one is the frequent systemic hemodynamic alteration, like blood pressure irregularities and a reduced systemic perfusion pressure in acute cardiac events [35]. The direct toxicity seems to be worse in patients with pre-existing renal insufficiency, diabetes mellitus, and atherosclerosis. Experimental studies showed, that after contrast medium exposure the renal blood flow and the glomerular filtration rate was reduced. This is due to a direct renal vasoconstriction effect of contrast medium [1], leading to ischemic kidney injury.

The incidence of a three vessel disease was higher in patients with NSTEMI than in patients with STEMI. We believe this finding to be attributable to an increased atherosclerotic burden in those patients. Montalescot et al. [21] demonstrated that patients with NSTEMI are older and have a greater amount of cardiovascular risk factors. This is in line to our findings. In addition to this, the contrast medium causes an increased diuresis through its osmotic effect leading to an intensified dehydration. This causes a decrease in renal blood flow and might pronounce the deleterious effect of contrast medium [8]. Furthermore almost 20% of these patients had a pre-existing renal insufficiency.

In contrast to this, patients with STEMI are younger and show less frequent cardiovascular risk factors except smoking which occurs more often in this group. These patients show a more severe clinical course including a cardiogenic shock. This systemic hemodynamic alteration leads to a reduced renal blood flow and could cause an acute renal impairment [17]. According to this, it is more complicated to differentiate between the toxic effect of the contrast agent and the acute renal failure through systemic hemodynamic impairment in STEMI patients.

In summary we could show for the first time, that there is no difference in developing CIN between NSTEMI and STEMI patients. However, we found several factors predisposing CIN independent from the underlying course of ACS like age (age >75 years), co-morbidity, hemodynamic impairment (LVEF <40% and cardiogenic shock) especially in STEMI patients and preexisting renal dysfunction (serum creatinine >1.5 mg/dl) mainly in NSTEMI patients. That leads to the hypothesis that hemodynamic alterations in the sense of prerenal impairment in STEMI and the atherosclerotic burden in the sense of renal failure in NSTEMI are the two different etiologies mainly underlying CIN in both groups of ACS patients. This points out the problem of differentiating between the toxic effect of contrast medium and systemic hemodynamic alteration leading to pre-renal kidney injury.

Apart from this, the development of CIN was associated with a significant longer in-hospital stay as well as a significantly higher mortality rate. In the multivariate analysis we could even show that CIN is an independent risk factor for increased in-hospital mortality—comparable with the development of a cardiogenic shock.

Study limitations

Although our study included more subjects than comparable studies, the relative limitations are those inherent in a single blind, single center study. Furthermore the group of patients who developed CIN is too small for a comparison between STEMI and NSTEMI with significant values. Thus, our findings should be confirmed in a larger multicenter trial. Furthermore, the definition of CIN is based on the absolute relative increase in Cr level, compared with baseline value, after a patient has been exposed to contrast agent, when alternative definitions have been excluded.

Conclusion

In times of acute PCI as state of the art therapy for ACS, CIN is a frequent and severe complication, even in patients without preexisting renal failure.

The results of this study show, that patients with NSTEMI and STEMI have a similar incidence of this complication although the underlying mechanisms might be different. Nevertheless, the mortality rate in patients with STEMI is more than two times higher. Especially in this group impaired hemodynamics seems to be an important cause not only for CIN. In contrast NSTEMI patients seem to suffer more from atherosclerotic burden as they present multivessel disease and preexisting renal dysfunction in a higher percentage.

This suggests that effort should be undertaken in early hemodynamic stabilization in STEMI patients whereas patients with NSTEMI could benefit more from a preventive strategy of renal protection like hydration before percutaneous intervention. This needs to be validated in a large prospective, randomized trial.

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