Risk factors of contrast-induced nephropathy in patients with acute coronary syndrome

Tomasz Rakowski[1], Artur Dziewierz[1], Michał Węgiel[1], Zbigniew Siudak[2], Wojciech Zasada[3], Jacek Jąkała[4], Dominika Dykla[5], Jerzy Matysek[4], Andrzej Surdacki[1], Stanisław Bartuś[1], Dariusz Dudek[1], Roman Wojdyła[3]

[1]Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland
[2]Collegium Medicum, Jan Kochanowski University, Kielce, Poland
[3]2nd Department of Cardiology and Cardiovascular Interventions, University Hospital, Kraków, Poland
[4]Krakow Center of Invasive Cardiology, Electrotherapy and Angiology, Kraków, Poland
[5]Center of Invasive Cardiology, Electrotherapy and Angiology in Nowy Sącz, Nowy Sącz, Poland

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Capodanno et al.

Correspondence to:
Tomasz Rakowski, MD, PhD, Institute of Cardiology, Jagiellonian University Medical College, Jakubowskiego 2, 30–688 Kraków, phone: +48 12 400 22 00, e-mail: mcrakows@cyfronet.pl

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ABSTRACT

Background: Patients with acute coronary syndrome (ACS) are at high risk of contrast-induced nephropathy (CIN), which is associated with prolonged hospitalization, higher morbidity and mortality after angiographic procedures. The occurrence of CIN is regarded as a transient and reversible condition. However, the persistence of CIN until hospital discharge in patients with ACS has not been thoroughly analyzed.

Aims: We aimed to analyze CIN persistent until hospital discharge in contemporary ACS population referred to invasive diagnostics and treatment.

Methods: A total of 2638 consecutive patients with ACS were included in a prospective registry. The occurrence of CIN was defined as a 25% increase in serum creatinine from baseline or a 0.5 mg/dl (44 µmol/l) increase in the absolute value.

Results: Criteria of CIN at hospital discharge were met in 10.7% of patients. Immediate percutaneous coronary intervention (PCI) after angiography (67% of patients) was associated with higher rates of CIN compared to patients referred for other treatment strategies (P <0.001). The logistic regression model showed that anemia at baseline (8.7% of patients) was an independent predictor of CIN, which occurred in 17.9% of anemic patients and 10% of patients without anemia (P <0.001). Also, ST-segment elevation myocardial infarction (STEMI) presentation and immediate PCI were independent predictors of CIN.

Conclusions: Despite intravenous fluid administration during the hospital stay, CIN persisted until hospital discharge in more than 10% of patients with ACS. Anemia at baseline, STEMI presentation, and immediate PCI strategy were independent predictors of CIN. Thus, preventive actions should be specially aimed at those groups of patients.

Key words: contrast-induced nephropathy, acute coronary syndrome, percutaneous coronary intervention, anemia

INTRODUCTION

Contrast-induced nephropathy (CIN) is an iatrogenic deterioration of renal function due to the injection of contrast media (CM) for angiography [1]. Patients with acute coronary syndrome (ACS) are at high risk of CIN development, which is associated with prolonged hospitalization and higher morbidity and mortality after angiographic procedures [2–4]. This invasive approach in patients with ACS improves outcomes but carries the risk of CIN. Patients presenting with ACS are

WHAT'S NEW?

Nowadays, in the era of frequent usage of contrast-based imaging and population aging (high-risk patients, multiple comorbidities), contrast-induced nephropathy (CIN) is a growing problem in clinical practice. In patients treated with primary percutaneous coronary interventions (PCI) for ST-segment elevation myocardial infarction (STEMI), contrast is usually administered without possibility of implementing CIN prevention or preprocedural information about renal function (lack of lab results before contrast administration), which may escalate CIN risks. In the presented registry, CIN was still present at discharge in more than 10% of patients with acute coronary syndromes and was more frequent in those treated with immediate PCI. STEMI presentation, immediate PCI, and anemia at baseline were independent predictors of CIN. Anemia was found to be not only a surrogate marker of renal function but also an independent predictor of CIN in our study. Thus, preventive actions, as well as careful monitoring, should be specially targeted at these groups of patients.

particularly susceptible to renal injury due to the frequent hemodynamic instability and lack of prophylaxis with optimal hydration before angiographic procedure due to indications for immediate interventions [5]. The occurrence of CIN is regarded as a transient and reversible condition. However, the persistence of CIN until hospital discharge in patients with ACS has not been thoroughly analyzed. Thus, we aimed to determine the predictors of CIN at the time of hospital discharge (persistent condition) in patients with ACS referred for coronary angiography.

METHODS

The presented study is an analysis based on an institutional registry prospectively collecting data of consecutive patients diagnosed with ACS and referred for coronary angiography in a single center. The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Institutional Review Board (OIL/KBL/57/2021). Consecutive ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina (UA) patients were included (Supplementary material, *Figure S1*). Patients treated with a conservative approach without coronary angiography were excluded from the study. Data on clinical characteristics, treatment strategies, renal function (serum creatinine level at baseline and during hospitalization), hemoglobin level (baseline and discharge), and in-hospital mortality were collected based on medical records. There was no independent angiographic analysis. Basic angiographic data were collected from procedure reports (operators' assessment). Serum creatinine levels at baseline and hospital discharge were compared. The occurrence of CIN was defined as a 25% increase in serum creatinine from baseline or a 0.5 mg/dl (44 µmol/l) increase in the absolute value. Glomerular filtration rate (GFR) was calculated according to the Modification of Diet in Renal Disease (MDRD) formula. Anemia at baseline was assessed based on blood sample analysis on hospital admission and was defined as hemoglobin <12 g/dl in women and <13 g/dl in men. For coronary angiography, an iso-osmolar, non-ionic CM has been used. During the hospital stay, patients underwent intravenous fluid administration according to routine local clinical practice.

Statistical analysis

Quantitative variables were described using means and standard deviation (SD; for normal distribution of data) or median with interquartile range (for non-normal distribution of data), where applicable. Categorical variables were presented as counts and percentages. The Mann-Whitney U test was applied for continuous variables. The χ^2 test was used for categorical (nominal and dichotomous) variables. Logistic regression models have been used to investigate predictors of CIN. All patient demographics, medical history, and procedural details were considered potential predictors. Then, the final models were constructed using forward selection with sex locked in the models. The level of statistical significance was set at an alpha value <0.05. The statistical analysis was performed using Statistica 13.3 software (Tibco Software Inc., Palo Alto, CA, US).

RESULTS

A total of 2638 consecutive patients with ACS undergoing coronary angiography (84.5% radial access) entered the study. Clinical data are demonstrated in Table 1. Most of the patients were male, and the most common clinical presentation was NSTEMI. Immediate percutaneous coronary intervention (PCI) after coronary angiography was performed in 1767 (67%) patients. Mean (SD) GFR at baseline was 94.1 (40.5) ml/min/1.73 m^2, and decreased baseline GFR (<60 ml/min/1.73 m^2) was found in 21% of individuals. Criteria of CIN at discharge were met in 10.7% of patients. Hospitalization length was slightly longer in the CIN group. Patients with CIN at discharge more often presented with STEMI diagnosis and underwent immediate PCI. They received more contrast during the procedure. Culprit lesion was found in the left main and/or left anterior descending coronary artery in 36.7% of patients (CIN vs. without CIN, 43.8% vs. 35.7%; *P* <0.001). Patent infarct-related artery (Thrombolysis In Myocardial Infarction [TIMI] grade 2 or 3 flow) on baseline angiography was less often present in patients with CIN

Table 1. Study group characteristics

	All patients (n = 2638)	CIN (+) (n = 281)	CIN (-) (n = 2357)	*P*-value
Male sex, n (%)	1704 (64.4)	175 (62.6)	1529 (64.6)	0.51
Age, years, median (IQR)	66 (58–76)	66 (57–76)	66 (58–76)	0.98
Previous MI, n (%)	683 (25.9)	53 (18.9)	630 (26.7)	0.02
Previous PCI, n (%)	488 (18.5)	35 (12.5)	453 (19.2)	0.01
Previous CABG, n (%)	84 (3.2)	8 (2.8)	76 (3.2)	0.6
Diabetes, n (%)	675 (25.6)	83 (29.5)	592 (25.1)	0.07
Active smoking, n (%)	886 (33.6)	95 (33.8)	791 (33.6)	0.68
Arterial hypertension, n (%)	2155 (81.7)	214 (76.5)	1941 (82.3)	0.045
Chronic kidney disease, n (%)	208 (7.9)	44 (15.7)	164 (7)	<0.001
Hb at baseline, g/dl, median (IQR)	13.7 (12.6–14.7)	13.4 (12.2–14.4)	13.7 (12.6–14.7)	0.01
Anemia at baseline, n (%)	229 (8.7)	41 (14.6)	188 (8)	<0.001
GFR <60 ml/min/1.73 m^2, n (%)	554 (21)	67 (24)	487 (20.6)	0.2
GFR <30 ml/min/1.73 m^2, n (%)	61 (2.3)	14 (5)	47 (2)	0.01
STEMI diagnosis, n (%)	958 (36.3)	131 (46.6)	827 (35)	<0.001
NSTEMI diagnosis, n (%)	1300 (49.3)	133 (47.3)	1167 (49.6)	0.49
UA diagnosis, n (%)	380 (14.4)	17 (6)	363 (15.4)	<0.001
Cardiogenic shock, n (%)	237 (0.9)	10 (3.6)	172 (0.6)	0.01
Femoral access, n (%)	340 (13)	51 (18)	289 (12)	0.008
PCI immediate after angiography, n (%)	1807 (68.5)	217 (77.4)	1590 (67.4)	0.001
Contrast media volume, ml, median (IQR)	200 (100–200)	200 (150–200)	200 (100–200)	0.01
Hospitalization length, days, median (IQR)	3 (3–4)	3 (3–4)	3 (3–4)	0.001

Abbreviations: CABG, coronary artery bypass grafting; GFR, glomerular filtration rate; Hb, hemoglobin; MI, myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction; UA, unstable angina

(36.7% vs. 49%; *P* <0.001). After PCI TIMI 3 flow was present in 94.8% of patients with CIN and 96.5% of patients without CIN (*P* = 0.3). Anemia at baseline was found in 8.7% of patients. Patients who underwent immediate PCI after angiography more frequently developed CIN than those referred for other treatment strategies (*P* <0.001). Similarly, patients with anemia at baseline (8.7% of patients) were at a higher risk of CIN than patients without anemia (*P* <0.001). There were no significant differences in rates of CIN between patients with decreased and normal (≥60 ml/min/1.73 m^2) baseline GFR (*P* = 0.2) (Figure 1). In patients with moderately decreased GFR (30–59 ml/min/1.73 m^2), the risk of CIN was 10.4%. When analyzing patients with severely decreased GFR (<30 ml/min/1.73 m^2), the CIN rate was higher in those patients compared to others (23.3% vs. 10.5%; *P* <0.01). The logistic regression model showed that anemia at baseline, age, immediate PCI after angiography, and STEMI presentation were the strongest independent predictors of CIN (Table 2). When analyzing a baseline Hb level instead of anemia as a categorical variable in the logistic regression model, the results were similar in terms of final variables included and their significance. The Hb level was independent predictor of CIN in this model (odds ratio [OR], 0.87; 95% confidence interval [CI], 0.79–0.94; *P* = 0.001). When analyzing STEMI vs. NSTEMI/UA patients, immediate PCI after angiography was performed in 90.1% and 64.6% (*P* <0.01). In patients with hospitalization time of 3 or more days (81% of patients), CIN at discharge was present in 11.6%, and similarly to the total cohort, the results were more frequent in patients with anemia at baseline (17.2% vs. 11%; *P* = 0.01). The rate of death during index hospitalization was higher in patients with CIN at discharge compared to those without CIN (2.1% vs. 0.3%; *P* = 0.001).

DISCUSSION

The most important findings from the presented analysis are (1) in patients with ACS undergoing invasive diagnostics and treatment, there is a high rate of persistence of CIN at the time of hospital discharge despite intravenous hydration during hospital stay; (2) anemia at baseline is a critical risk factor of CIN at discharge. Other important factors are STEMI presentation and immediate PCI strategy.

Definition of CIN varies among individual studies. The most common definition of CIN is a 25% increase in serum creatinine from baseline or a 0.5 mg/dl (44 µmol/l) increase in absolute value within 3 days following the intravascular administration of CM [6]. Typically, CIN is a self-limiting process with serum creatinine levels peaking in 3–5 days and usually returning to baseline values 7–10 days after angiographic procedures [1, 7]. In the presented analysis, we tried to investigate the rate of persisting kidney injury until hospital discharge, and we demonstrated that CIN is not only a transient in-hospital complication but might also affect kidney function after discharge. Unfortunately, no specific treatment for CIN is available. Thus, most actions aim at prevention [8]. Over the years, several strategies have been investigated for CIN prevention including N-Acetyl-L-Cysteine, statins, and ascorbic acid administration. However, consistent data showing their efficacy are still lacking [9, 10]. Some trials showed the beneficial role of statins in reducing the risk of CIN, yet other studies did not support that claim [11–13]. Simple fluid administration remains the

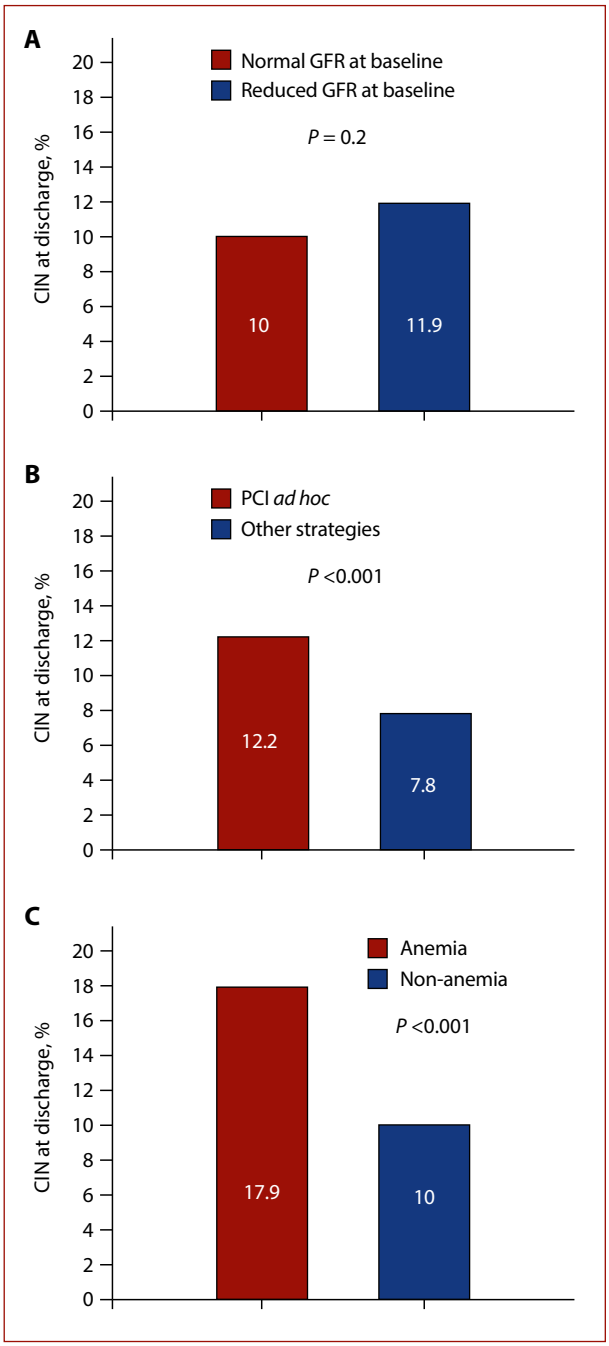

Figure 1. Contrast-induced nephropathy rate at discharge according to GFR at baseline (**A**), revascularization strategy after angiography (**B**), and anemia at baseline (**C**)

Abbreviations: CIN, contrast-induced nephropathy; other — see Table 1

Table 2. A logistic regression model to investigate predictors of CIN at discharge

Variable	OR (95% CI)	*P*-value
Anemia	2.14 (1.43–3.21)	<0.001
Age (per 1 year)	1.02 (1.01–1.04)	0.002
STEMI (vs. NSTEMI + UA)	1.69 (1.31–2.20)	<0.001
Immediate PCI strategy	1.39 (1.01–1.9)	<0.001
Female (vs. male)	1.1 (0.83–1.46)	0.57
GFR	1.01 (1.01–1.01)	<0.001

Abbreviations: see Table 1

most effective strategy for CIN prevention [14, 15]. Volume expansion prevents intrarenal hemodynamic instability, minimizing vasoconstriction and medullary ischemia. Dilution of CM by more fluid reduces its concentration and nephrotoxicity [16]. Also, some techniques were proposed to reduce contrast volume during the intervention [17].

Patients presenting with ACS are particularly susceptible to CIN development due to the necessity for urgent angiographic procedures (especially STEMI and very high-risk NSTEMI), without the possibility of achieving optimal hydration before CM injection or prior to withdrawal of potentially nephrotoxic drugs [5, 14]. Hemodynamic instability of these patients often causes administration of loop diuretics and hampers transfusion of a larger amount of fluid, which increases medullary ischemia [17]. In our registry, immediate PCI after coronary angiography was associated with higher CIN rates, which might be linked to a larger amount of CM being injected during a one-step procedure. Also, we found STEMI presentation (need for immediate invasive strategy) as an independent CIN predictor.

In our study, anemia at baseline was an independent predictor of CIN. Nikolsky et al. [19] showed that each 3% decrease in baseline hematocrit resulted in a significant increase in the odds of CIN after PCI. Similarly, Sreenivasan et al. [20] described a growing risk of CIN with increasing severity of anemia. A possible explanation of that mechanism is an aggravating role of anemia in renal ischemia [21, 22]. Injection of CM triggers medullary hypoxic injury by increasing local renal vascular resistance [23]. A combination of exposure to CM and low hemoglobin decreases delivery to the outer medullary region, which is particularly susceptible to ischemic injury. Theoretically, preventive red-blood cell transfusion before angiographic procedures might reduce the risk of CIN in those patients. However, there are no scientific data to support this hypothesis. Also, the level of hemoglobin on admission is frequently not known in ACS patients, especially in those with STEMI. Thus, preventive red-blood cell transfusion might not be an option for patients with ACS requiring immediate coronary angiography. Besides coronary interventions, acute kidney injury is an important problem during complex percutaneous cardiovascular interventions like transcatheter aortic valve implantation (TAVI). Dedicated studies have described predictors of this complication in patients undergoing TAVI [24].

Our study has some limitations. This is a single-center registry analyzing in-hospital course, and follow-up data on renal function and clinical outcomes are not available. During hospitalization, patients underwent intravenous hydration. However, detailed individual patients' data was not recorded. The length of hospital stay was not equal for all patients, which affects the time interval for CIN development and assessment. Some data like the hemodialysis rate, pharmacotherapy, and long term follow-up are lacking. Angiographic data are based on procedure reports but not on independent angiographic analysis. On the other

hand, the study represents real-life data with consecutive patients enrolled.

CONCLUSIONS

Presented analysis shows that CIN was still present at discharge in more than 10% of patients with ACS despite intravenous fluid administration during the hospital stay. Anemia at baseline, STEMI presentation, and immediate PCI strategy were independent predictors of CIN. Thus, preventive actions, as well as careful monitoring, should be specially targeted at these groups of patients.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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