Gavrilko A. D.[1], Mezhonov E. M.[2], Shalaev S. V.[2], Abdullaev D. E. Ogly[2], Shermuk A. A.[2], Kuslivyi A. M.[2], Krasheninin D. V.[2], Vyalkina Yu. A.[1]

[1] Tyumen State Medical University, Tyumen, Russia
[2] Regional Clinical Hospital #1, Tyumen, Russia

Influence of Loading Dose Of Atorvastatin on the Risk of Contrast-Induced Nephropathy in Patients With ST-Segment Elevation Myocardial Infarction

Aim	This retrospective cohort study focused on evaluating the incidence of contrast-induced nephropathy (CIN) associated with administration of an atorvastatin loading dose (80 mg) prior to invasive coronary angiography (CAG) in patients with ST-segment elevation myocardial infarction (STEMI).
Material and Methods	This retrospective cohort study included 386 patients with STEMI. The patients were divided into two groups: intervention group (n=118) and control group (n=268). Patients in the intervention group, at the stage of admission to the catheterization laboratory, were administered a loading dose of atorvastatin (80 mg, p.o.) immediately before access (introducer placement). The endpoints were development of CIN, which was determined by increased serum creatinine 48 h following the intervention by at least 25% (or 44 μmol/l) of baseline value. In addition, in-hospital mortality and incidence of CIN resolution were assessed. To adjust the groups for dissimilar characteristics, a «pseudorandomization» method was used by comparing propensity scores.
Results	The incidence of CIN was significantly lower in the intervention group than in the control group (10.5% vs. 24.4%; p=0.016) with the odds for the CIN development lower than in the control group (odds ratio (OR) 0.36; 95% confidence interval (CI), 0.16–0.85). Creatinine concentrations returned to the baseline value in 7 days more frequently than in the control group (66.3% vs. 50.6%, respectively; OR, 1.92; 95% CI, 1.04–3.56; p=0.037). In-hospital mortality was higher in the control group but did not differ significantly between the groups.
Conclusion	Administration of atorvastatin 80 mg to STEMI patients immediately before CAG was associated with a reduced risk of CIN and a higher likelihood of serum creatinine returning to the values at admission by day 7.
Keywords	Contrast-induced nephropathy; ST-segment elevation myocardial infarction; atorvastatin; percutaneous coronary intervention
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Corresponding Author	Gavrilko A.D. E-mail: dalmatov.artem@gmail.com

Introduction

Contrast-induced nephropathy (CIN) implies renal dysfunction developing within 3 days after intravascular administration of radiocontrast agents if there is no alternative etiology [1]. CIN is diagnosed by elevated serum creatinine by >25% or >44 μmol/L within 48–72 hours after the administration of a radiocontrast agent [2, 3]. This definition is the most common in the literature, which makes it possible to perform a reliable comparison between numerous studies, in which the definition of CIN was used. Although CIN-associated elevated creatinine rarely requires renal replacement therapy (RRT) in most cases [4], the presence of acute kidney injury (AKI), in which CIN is conventionally considered, is associated with an increased risk of adverse outcomes [5, 6]. RRT is the only effective treatment for CIN. This determines the high relevance of the search for CIN risk factors and the development of new preventive measures. The primary preventive measures for CIN include pre-hydration and post-hydration with isotonic sodium chloride solution, minimizing the administered volume of the radiocontrast agent, using radial access in percutaneous coronary intervention (PCI), discontinuing nephrotoxic drugs a few days prior to the intervention with the use of the radiocontrast agent [7, 8]. Patients with ST-segment elevation myocardial

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infarction (STEMI) represent the most vulnerable group from this perspective due to the inability to postpone emergency intervention in order to identify CIN risk factors and take out preventive measures. Thus, it is necessary to ensure that preventive measures are available in a PCI facility, which would not delay the implementation of invasive coronary artery angiography (CAG) and would not require complex and/or specific actions from health professionals. There is numerous evidence that statin therapy prior to endovascular interventions reduces the likelihood of periprocedural MI [9]. The pleiotropic effect of statins due to their anti-inflammatory and antioxidant properties created the prerequisites for their use for CIN prevention before conducting examinations with the use of radiocontrast agents [10]. The hypothesis that statins reduce the risk of this complication was confirmed in a meta-analysis of 124 studies (28,240 patients) comparing the 10 best-studied CIN prevention strategies [11]. However, only few studies are devoted to patients with STEMI. Such studies also have strict inclusion and exclusion criteria, which, in our opinion, limits the possibility of applying their findings for all patients hospitalized the PCI facility with the diagnosis of STEMI.

Objective

Estimate of the frequency of CIN after loading dosing of atorvastatin (80 mg) before CAG in patients with STEMI, the frequency of serum creatinine recovery to baseline on day 7 after the invasive intervention, and hospital mortality rate.

Material and methods

The retrospective cohort study included 386 patients with STEMI. The study was conducted from 2016 to 2021 in Tyumen Regional Clinical Hospital no.1. Patients were supervised from admission to discharge from the hospital.

Inclusion criteria:

1) STEMI diagnosed by the clinical picture and electrocardiogram;
2) indications for emergency CAG given the current clinical guidelines.

Exclusion criteria:

1) acute hepatitis;
2) pregnancy;
3) death in the emergency room, during transfer to the X-ray surgery room, within the first 2 days after the intervention.

Patients of the intervention group (n=118) received a loading dose of atorvastatin (80 mg orally) immediately before the access provision (insertion of a sheath introducer) when they were delivered in the X-ray surgery room.

Patients of the control group (n=268) did not receive a loading dose of statins before the intervention.

Patients did not receive pre-hydration. Post-procedural hydration (400 mL of 0.9% sodium chloride solution intravenously) was recommended for all patients except for patients with acute heart failure (Killip≥III). Iohexol 350 mg/mL was used as the radiocontrast agent in all cases. CAG, revascularization and further medications were carried out following the standard protocol in accordance with the current Russian guidelines and the guidelines of the European Society of Cardiology [12]. During the observation, the following parameters were evaluated: clinical and anamnestic parameters at admission, left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), left ventricular ejection fraction (LVEF), the presence of LV aneurysm in echocardiogram recorded the day after the intervention, the peak level of troponin I, creatine phosphokinase MB (CPK-MB) within the period before the repeated analysis for creatinine, hemoglobin and cholesterol levels at admission, angiographic indicators evaluated before and after stent implantation (Table 1).

The endpoint was the fact of developing CIN, which was established by elevated levels of serum creatinine 48 hours after the intervention by at least 25% (or 44 μmol/L) of the baseline [10]. Moreover, hospital mortality, serum creatinine, and glomerular filtration rate (GFR; using CKD-EPI formula) were estimated on day 7 after the intervention. The recovery of serum creatinine to the baseline levels registered at admission were also estimated to determine the frequency of AKI resolution. A decrease in creatinine to levels below the baseline at admission of +10% on day 7 was considered positive [13]. The study protocol was approved by the local ethics committee of the Tyumen State Medical University (Russian Federation).

The data obtained were process in Jamovi v. 1.6.16.0 and SPSS v. 23.0. Parametric (Student's t-test, Welch t-test) and non-parametric (Mann–Whitney test) methods were used depending on the type of indicator distribution in the samples. The chi-squared test and Fisher's exact test were used for the categorical variables. Normally distributed data are presented as M±SD, where M is arithmetic mean and SD is the standard deviation, non-normally distributed data are expressed as the medians and an interquartile ranges (Me [25th percentile; 75th percentile]). The pseudorandomization method by comparing propensity scores was used to align the compositions of the groups by the charac-

teristics with statistically significant baseline differences and to remove the effects of the detected differences on the frequency of the outcomes of interest. The pseudorandomization sample was used to evaluate the endpoints. Odds ratios (OR) and 95% confidence intervals (CI) were calculated for qualitative endpoints. OR was used as a quantitative measure of the effect when comparing relative indicators. It was defined as the ratio of the probability of an event in the group exposed to the risk factor to the probability of an event in the control group. The differences were considered statistically significant with a two-tailed level of significance $p<0.05$.

Results

Of the 386 patients included in the study, 99 (25.6%) were female. Mean age was 59±9.87 years. CAG was not performed in 6 patients in the control group due to refusal of the intervention, 14 patients did not undergo PCI (2 in the intervention group and 12 in the control group). PCI was not performed for the following reasons:

1. Patient did not have a hemodynamically significant coronary artery involvement (n=1)
2. Patient had indications for emergency coronary artery bypass grafting (n=6);
3. Failure to conduct an endovascular instrument to the target zone due to anatomical variations (n=1);
4. Chronic occlusion in the infarct-related artery (n=6).

Patients of the intervention group were younger (p=0.027), had higher levels of GFR (p=0.004), LVESV (p=0.011), troponin I (p=0.016), CPK-MB (p=0.011), hemoglobin (p=0.011), a higher rate of LV aneurysm (p=0.02), and a history of kidney disease (p<0.001), and a lower rate of anemia (p<0.001), recurrent MI (p<0.001), and lower LVEF (p<0.001).

New groups were formed after pseudorandomization, with 86 patients in each. The characteristics of the groups are provided in Table 1.

Thus, the study groups were comparable after pseudorandomization in age, GFR, LVEF, LVESV, hemoglobin, CPK-MB, and troponin I levels, and the incidence of recurrent MI, LV aneurysm, a history of kidney disease, and anemia. Moreover, pseudorandomization excluded 6 patients who refused from CAG, and the number of patients who did not undergo PCI decreased to 4 (2 in each group).

GFR was statistically significantly higher in the intervention group on day 7: 87 mL/min versus 75.5 mL/min (p=0.008). Serum creatinine levels were also statistically significantly different and statistically significantly lower in the intervention group on day 7: 82.5 μmol/L versus 89.5 μmol/L (p=0.018; Table 2).

Table 1. Characteristics of patient groups (after pseudorandomization)

Parameter	Statins – (n=86)	Statins + (n=86)	p
Age, years, M±SD	61.1±12.3	59.5±9.4	0.330
Female, n (%)	16 (18.6)	16 (18.6)	1.0
Creatinine, μmol/L	79 [72; 91]	80.5 [68.3; 95]	0.910
GFR, mL/min/1.73m²	84.5 [70; 95]	87.5 [72; 101]	0.410
Radiocontrast agent, mL	100 [90; 140]	115 [100; 200]	0.658
LVEF, %	54.4 [47; 61]	52.7 [45; 60]	0.211
LVEDV, mL	132 [115; 150]	130 [114; 145]	0.678
LVESV, mL	55 [48; 70]	58 [45; 79.3]	0.157
Troponin I, ng/mL	0.72 [0.25; 1.41]	0.92 [0.39; 2]	0.142
CPK-MB, U/L	34.6 [22.4; 46.8]	104 [68.5; 260]	0.06
Hemoglobin, g/L	141 [130; 151]	145.5 [135; 151]	0.490
Cholesterol, mmol/L (M±SD)	4.77±1.33	4.7±0.98	0.694
Statin therapy before hospitalization, n (%)	20 (23.3)	18 (20.9)	0.713
Symptom-to-balloon time, min, (Me±SD)	398±23	355±46	0.4
Anterior MI, n (%)	40 (46.5)	29 (33.7)	0.09
Three-vessel involvement, n (%)	18 (20.9)	23 (26.7)	0.371
Thrombolysis, n (%)	15 (17.4)	15 (17.4)	1.0
Blood flow TIMI<2 before the intervention, n (%)	55 (64)	46 (53.5)	0.163
Blood flow TIMI<2 after the intervention, n (%)	2 (2.3)	2 (2.3)	1.0
Killip>II, n (%)	3 (3.5)	7 (8.1)	0.192
Killip III–IV, n (%)	1 (1.2)	6 (7)	0.117
Anemia*, n (%)	17 (19.8)	13 (15.1)	0.422
Recurrent MI, n (%)	18 (20.9)	15 (17.4)	0.561
LV aneurysm, n (%)	10 (11.6)	11 (12.8)	0.816
Arterial hypertension, n (%)	80 (93)	72 (83.7)	0.094
Diabetes mellitus, n (%)	12 (14)	12 (14)	1.0
Chronic obstructive pulmonary disease, n (%)	3 (3.5)	3 (3.5)	1.0
History of kidney disease, n (%)	7 (8.1)	8 (9.3)	1.0
IABP, n (%)	1 (1.2)	1 (1.2)	1.0

* Erythrocytes<4.0 mln/μL, hemoglobin <130 g/L in male patients; erythrocytes<3.8 mln/μL, hemoglobin<120 g/L in female patients. GFR, glomerular filtration rate; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; CPK, creatine phosphokinase; IABP, intra-aortic balloon pump counterpulsation.

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When the study groups were aligned, the incidence of CIN was statistically significantly lower in the intervention group than in the control group (Table 3). Creatinine levels recovered to the baseline after 7 days more often in the intervention group compared to the control group. Hospital mortality was comparable in the control group and the intervention group, although there was a trend to higher rate of the control group (Table 3).

Discussion

The main finding of our study was that STEMI patients who received atorvastatin 80 mg in the X-ray room immediately before CAG had a lower incidence of CIN than patients who did not receive a loading dose of the statin before the invasive intervention. Patients of the intervention group were also more likely to have serum creatinine recovered to the baseline levels (at admission) by day 7 of the observation. There was a trend to lower mortality in the group of patients who received atorvastatin. This rate was significantly lower than in the general population of STEMI patients. Initially, there were 3 in-hospital deaths in the intervention group, which was 2.5%, but 2 of 3 patients were withdrawn from the study during pseudorandomization. However, this figure is still much lower than in the general population of STEMI patients.

There could be two causes for this. First, we assume that preoperative mortality was possible in the emergency room or during transportation to the X-ray surgery room. Thus, those patients could have affected total mortality but were not included in the study and therefore, that could have determined lower mortality rates in our study. Second, 3 patients died in the intensive care unit before repeated blood test to establish the development of CIN. Those patients were not included in the study following the exclusion criteria.

A large amount of literature data has been accumulated that confirm the efficacy of statin therapy in preventing CIN before and immediately after radiocontrast interventions [14, 15]. However, there is a few researches on the efficacy of such a prevention strategy in STEMI patients. Our findings are consistent with the results of a randomized placebo-controlled study by Li et al. [16] who investigated the effect of the loading dose of atorvastatin 80 mg followed by the long-term administration of atorvastatin 40 mg in patients with STEMI. Our study has some differences from the above. First, Li et al. used more strict inclusion and exclusion criteria. The study did not include patients who had received statins before admission to the hospital, had a history of kidney disease, uncontrolled arterial hypertension, received thrombolytic therapy, and patients with cardiogenic shock who required intra-aortic balloon pump counterpulsation. The study included only patients with STEMI hospitalized within the first 12 hours. Such criteria significantly limit the possibility of extrapolating the results to all patients with STEMI. Our study did not have such strict selection criteria in terms of the severity of patient's condition, the duration of hospitalization, the presence of kidney disease, and a history of thrombolysis. Thus, despite the same CIN criteria, the incidence was higher in our study than in the study by Li et al. (10.5% versus 2.6% in the intervention groups and 24.4% versus 15.7% in the control groups), which can be explained by looser selection criteria in our study. Our findings are also consistent with the results of a randomized study by Leoncini et al. [17] who evaluated the effect of high-dose rosuvastatin (40 mg at admission) in patients with NSTEMI prior to the endovascular intervention. The exclusion criteria in this study were statin therapy before the development of ACS, very high risk at admission requiring CAG within less than 2 hours, and end-stage chronic kidney disease or

Table 2. Outcomes (quantitative endpoints)

Parameter	Statins – (n=86)	Statins + (n=86)	p
Creatinine in 7 days, μmol/L	89.5 [79; 101]	82.5 [71.3; 92.5]	0.018
GFR in 7 days, mL/min	75.5 [62; 88.8]	87 [69.1; 97.8]	0.008

GFR, glomerular filtration rate.

Table 3. Outcomes (qualitative endpoints)

Parameter	Statins – (n=86)	Statins + (n=86)	p	OR (95% CI)
CIN. n (%)	21 (24.4)	9 (10.5)	0.016	0.36 (0.16–0.85)
Creatinine level recovery to baseline on day 7. n (%)	43 (50.6)	57 (66.3)	0.037	1.92 (1.04–3.56)
In-hospital mortality. n (%)	6 (7.0)	1 (1.2)	0.120	0.17 (0.02–1.47)

OR. odds ratio; CI. confidence interval; CIN. contrast-induced nephropathy.

serum creatinine>265 μmol/L. As in the study by Li et al., patients of the intervention group in the study by Leoncini et al. took statins after the intervention according to the study protocol (atorvastatin 40 mg for 48 hours twice a day in the study by Li et al. and rosuvastatin 20 mg continuously once a day in the study by Leoncini et al.). In our study, post-intervention medication was not regulated and was determined by the attending physician given the current guidelines; the frequency of statin administration after the invasive intervention was comparable between the groups: 99% and 98% (p=0.6750), and doses did not differ: 60 [40; 80] mg and 60 [35; 80] mg (p=0.563). It can be assumed from the literature that statins have the most pronounced protective effect in patients who did not take them before the development of STEMI, since almost all studies excluded patients who took statins before MI. Our study included 23.3% and 20.9% of patients who took statins prior to hospitalization in the control and intervention groups, respectively.

In our study, the preventive effect of atorvastatin at a dose of 80 mg was traced in a wider population of STEMI patients without statin therapy prescribed by the protocol after the intervention. The study was limited by the lack of true randomization, which makes possible the presence of confounding factors that may contribute to the endpoints apart from the fact of the administration of atorvastatin in the intervention group. Moreover, we did not take into account the daily doses of statins administered before hospitalization, which could potentially affect the risk of developing CIN.

Conclusion

The use of atorvastatin at a dose of 80 mg immediately prior to invasive coronary angiography in patients with ST-segment elevation myocardial infarction is associated with a lower risk of contrast-induced nephropathy. The use of atorvastatin before the intervention is associated with higher likelihood of the recovery of serum creatinine to the baseline values by day 7 of hospital treatment.

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