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High dose of Atorvastatin versus Rosuvastatin as Preventive Strategies for Contrast-Induced Acute Kidney Injury in Patients Undergoing Percutaneous Coronary Intervention

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ORIGINAL ARTICLE

Atorvastatin versus Rosuvastatin as Preventive Strategies for Contrast-Induced Acute Kidney Injury in Patients with Percutaneous Coronary Intervention

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ABSTRACT

Background and Objective: The huge assortment of accessible similar preliminaries gave very opposing ends that has made it hard to find out the best methodology for Contrast-induced acute kidney injury (CI-AKI) anticipation in clinical practice. So this investigation meant to find compelling preventive system for CIAKI through assessing the adequacy of rosuvastatin and atorvastatin after PCI. **Methods**:Non-Randomized controlled trail of patients experiencing PCI done at Zagazig University and Ahmed Maher Teaching Hospital. Subjects were arranged into 3 goups : group I incorporate 79 patients getting 40 mg rosuvastatin ,group II incorporate 79 patients getting 80 mg of atorvastatin ultimately group III 79 patients who didn't get high stacking portion of statins before essential PCI. **Results**: The rate of CIN was essentially higher among control group when contrasted against those getting statin stacking portion. Besides, CIN was related to age > 60 years, hypertension or DM , those patients with EF <50 and HB level ≤ 12 , those had dyslipidemia and their baseline Serum creatinine > 1 mg/dl ($p<0.01$). **Conclusion**: Rosuvastatin pretreatment applies an impact like atorvastatin in forestalling CIN in patients experiencing PCI.

Keywords: CI-AKI, coronary angiography, serum creatinine and PCI, intense kidney damage.

INTRODUCTION

Contrast-induced acute kidney injury (CI-AKI) is a known complication of intravascular administration of contrast media used in coronary angiography (CAG) and percutaneous coronary interventions (PCI) [1]; and is associated with increased mortality, morbidity, healthcare expenditure, and prolonged hospital stay. CI-AKI has become the third leading cause of iatrogenic renal failure in the United States [2]. Previous report indicated that even mild postoperative AKI is independently associated with an almost 5-fold increase in in-hospital death [3]. Clinically, the rate of CI-AKI is more noteworthy in patients with cardiovascular illnesses or previous renal insufficiency [4]. The detailed rate ranges from 2 to 50%. Hydroxy-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins) are intense inhibitors of cholesterol biosynthesis and apply beneficial impacts in the essential and optional avoidance of coronary disease (CAD)[5].

The prophylactic benefit of statins in diminishing the occurrence of CI-AKI has been researched in a few observational and randomized studies [6,7,8]; be that as it may, different examinations have revealed

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inconsistent and conflicting results [9,10]. Besides, regardless of whether preoperative statin treatment has a preventive, impartial, or hindering job on AKI stays hazy and fervently discussed. Supposedly, no studies have assessed the connection between high plasma presentation of statins and the danger of CI-AKI. Along these lines, the point of the present investigation is to think about the viability of Atorvastatin versus Rosuvastatin in a high portion for forestalling AKI in Patients experiencing PCI .

SUBJECTS AND METHODS

Study structure:

Non-randomized controlled clinical study was conducted in cardiology Department, Faculty of medicine, Zagazig University and Ahmed Maher Teaching hospital. Approval for the study was obtained from International Review Board **(IRB),** Faculty of Medicine- Zagazig University. 237 adult subjects were included in it (79 in each group). Informed written consents were obtained from all of them to use their samples and clinical data in this study according to the Declaration of **Helsinki** using a dedicated form.

Inclusion and exclusion criteria:

Patients conceded for PCI with ordinary kidney capacities and serum creatinine; Meanwhile, patients with a high-hazard highlights justifying emergency coronary angiography (within 2 hours) those with Cardiogenic shock or post arrest as a result of MI , acute renal failure or end-stage renal disease requiring dialysis , serum creatinine >3 mg/dL in a CKD patient , contrast medium administration during the previous 10 days and absence Laboratory information including serum creatinine were excluded.

Operational design:

Subjects was characterized into 3 groups as following: Group I: Patients experienced PCI getting high portion (40mg) of Rosuvastatin, II : Patients experienced PCI accepting 80mg Atorvastatin and Group III: Control group undergoing PCI without receiving high dose Atorvastatin or Rosuvastatin.

All subjects experienced the accompanying: full history; including family history and pervious treatment, Complete physical and clinical assessment and blood sampling for getting serum tests for estimating: Serum creatinine (SCr) upon admission and inside 48–72 h after PCI exposure,Blood urea nitrogen (BUN) ,Creatine kinase MB and Fasting glucose.

Percutaneous coronary intervention

Aspirin (300 mg) and clopidogrel (600 mg) were loaded in all patients before the procedure. An intravenous bolus of 5000 U unfractionated heparin was given to keep up activated coagulating time >300 seconds during the procedure. Coronary angiography and stent implantation were performed utilizing standard interventional techniques. Platelet glycoprotein IIb/IIIa inhibitor was administrated in some elective cases. Aspirin (100 mg/day), clopidogrel (75 mg/day), and statins were recommended to all patients after the procedure. Hydration treatment (0.9%NaCl, 1 mL/kg/h) was performed during the pre-and post-PCI periods and diminished to 0.5 mL/kg/h for patients with a left ventricular ejection fraction (EF) <40%.

STATISTICAL ANALYSIS

Data were check ,entered and analysed by using SPSS version 24. Continuous variables were expressed as mean +- SD or median ,and categorical variables as frequency and percentage .Student t-test , ANOVA test , Chi-squared test , Wilcoxon rank sum test and Mann-Whitny were used when appropriate . Logestic regression analysis was performed using CIN as the dependent variable .Variables that were statistically significant according to a univariate analysis were included in the final multivariate model to identify CIN predictors.

RESULTS

Our study showed that the studied group were comparable as regard demographic data ,age and gender distribution. The clinical characteristics were not statistically significant, the proportion of hypertension and DM were comparable among studied group (p=0.8 and p=0.53 respectively).The studied groups were

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also similar as regard level of blood pressure and pulse rate **(Table 1)**.

Our results revealed that the only significant change in S.cr. in comparison to baseline was found among the control group (not receiving loading dose of statin) at 48 h and 72 h while there were no statistical significant observed among groups or within each group of Rosuvastatin or Atorvastatin **(Table 2).**

According to the incidence of contrast-induced nephropathy, our results showed that, the incidence of CIN was significantly higher among control group when compare with those receiving statin loading dose **(Table 3)**.

Regarding comparison between Rosuvastatin and Atorvastatin in serum creatinine and incidence of contrast induced nephropathy, our results revealed that pretreatment loading dose with Rosuvastatin had a similar effect as Atovastatin regarding the development of CIN in patient undergoing PCI . The incidence of CIN was 5.1% in Rosuvastatin compared to 6.3% in Atorvastatin (p=0.73) **(Table 4).**

Additionally, our results showed that CIN was associated with age > 60 years, hypertension or DM , those patient with EF <50 and HB level ≤ 12 , those had dyslipidemia and their baseline Serum creatinine > 1 mg⁄dl (p<0.01) , while gender or CRP were not statistically significant **(Table 5).**

Table (1): Demographic characteristics and medical history.

	(I) Rousuvastatin n = 79	(II) Atorvastatin n = 79	(III) Control n = 79	F	P
Age (years)					
$\bar{X}$±SD	57.3±9.2	56.9±9.0	55.9±8.2	0.5	0.58
Range	40-75	40-75	42-72		
Gender				X^2	P
Male N (%)	53 (67.1%)	53 (67.1%)	47 (59.5%)	1.33	0.51
Female N (%)	26 (32.9%)	26 (32.9%)	32 (40.5%)		
Hypertension N(%)	24 (30.4%)	26 (32.9%)	27 (34.2%)	0.27	0.8
Diabetes mellitus N(%)	26 (32.9%)	20 (25.3%)	25 (31.6%)	1.25	0.53
SBP (mmHg)				F	P
$\bar{X}$±SD	126.2±25	128±22	125±21.9	0.4	0.6
Range	90-180	90-180	90-180		
DBP (mmHg)					
$\bar{X}$±SD	78.9±14	80.2±14	78.3±14.3	0.5	0.58
Range	60-120	60-120	60-120		
Heart rate (bpm)					
$\bar{X}$±SD	85.2±9.8	86.4±9.1	85.3±8.7	0.41	0.65
Range	60-110	60-110	60-110		

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Table (2): Changes in serum creatinine (Scr) among studied groups

	I n = 79	II n = 79	III n = 79	F	P
Baseline					
$\bar{X}$±SD	1.0±0.3	0.98±0.29	0.98±0.27	0.06	0.9
Range	0.5-1.8	0.5-1.8	0.5-1.7		
24 hours					
$\bar{X}$±SD	1.125±0.35	1.09±0.4	1.125±0.4	0.16	0.85
Range	0.54-2.2	0.54-2.7	0.54-3.0		
48 hours					
$\bar{X}$±SD	1.1±0.36	1.11±0.31	1.17±0.37*	0.5	0.59
Range	0.55-2.23	0.55-2.4	0.6-2.3		
72 hours					
$\bar{X}$±SD	1.13±0.3	1.14±0.4	1.2±0.35*	0.2	0.8
Range	0.52-2.24	0.52-2.65	0.52-2.4		

Table (3): Incidence of contrast-induced nephropathy

	I		II		III		X^2	P
	N	%	N	%	N	%		
Contrast induced nephropathy								
No	75	94.9	74	93.7	66	83.5	7.3	0.02* Sig
Yes	4	5.1	5	6.3	13	16.5		

Table (4): Comparison between Rosuuvastatin and Atorvastatin as regard changes in serum creatinine and incidence of contrast induced nephropathy

	I n = 79	II n = 79	T	P
Changes in Scr Baseline				
$\bar{X}$±SD	1.0±0.3	0.98±0.29	0.34	0.72
Range	0.5-1.8	0.5-1.8		
24 hours				
$\bar{X}$±SD	1.1±0.35	1.09±0.4	0.23	0.8
Range	0.54-2.2	0.54-2.7		
48 hours				
$\bar{X}$±SD	1.125±0.36	1.11±0.31	0.24	0.8
Range	0.55-2.23	0.55-2.4		
72 hours				
$\bar{X}$±SD	1.13±0.3	1.14±0.4	0.05	0.95
Range	0.52-2.24	0.52-2.65		
% of changes Scr			0.8	0.42
$\bar{X}$±SD	12.5±9.14	13.7±9.6		
Range	0.0-41	0.0-41		
			X^2	P
Incidence of CIN N (%)	4 (5.1%)	5 (6.3%)	0.12	0.73

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Table (5): Factors associated with CIN after PCI.

	Without CIN N = 215		With CIN N = 22		X^2	P
	N	%	N	%		
Age (years)						
≤ 60	150	70.2	5	22.7	20.0	<0.001**
> 60	64	29.8	17	77.3		
Gender						
Male	131	60.9	12	54.5	0.75	0.38
Female	84	39.1	10	45.5		
Hypertension	62	28.8	15	68.2	14.09	0.001**
Diabetes mellitus	59	27.4	12	54.5	6.99	0.008**
EF						
< 50	70	32.6	14	63.6	8.42	0.003**
≥ 50	145	67.4	8	36.4		
CRP						
≤ 4	29	13.5	2	9.1	0.34	0.56
> 4	186	86.5	20	90.9		
HB						
≤ 12	82	38.1	18	81.8	15.6	0.001**
> 12	133	61.9	4	18.2		
Dyslipidemia						
Yes	53	24.7	13	59.1	11.7	0.001**
No	162	75.3	9	40.9		
Baseline Scr						
≤ 1.0	147	68.4	6	27.3	14.73	<0.001**
> 1.0	68	31.6	16	72.7		

DISCUSSION

Over the past 25 years, the number of percutaneous procedures requiring contrast media administration has increased exponentially Contrast-induced acute kidney injury (CIAKI) is not an infrequent complication of coronary angiography and percutaneous coronary intervention and has been associated with increased mortality and cardiovascular events [11,12]. The optimal CIAKI prevention strategy for patients with suspected or confirmed coronary artery disease undergoing percutaneous coronary procedures is unknown. A wide array of medications and hydration regimens have been investigated in recent years.

The studied groups were comparable as regard demographic data, age and gender distribution. The clinical characteristics were not statistically significant, the proportion of hypertension and DM were comparable among studied groups (p=0.8 and p=0.53 respectively). From the present study the incidence of CIN was significantly higher among control group when compared with those receiving statin loading dose .The prevention of CIN is an important concern because it affects patient morbidity and mortality. In the current study, we found that the incidence of CIN was 5.1% in group I , 6.3% in group II and 16.5% in group III, in agreement with **Tsai et al.,** [13] .. Few strategies have been demonstrated to be effective for preventing CIN. This has led to an increased interest in the preventive effects of statins (especially, atorvastatin and rosuvastatin) on CIN development in patients undergoing PCI. However, conflicting results have been

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published. **Kandula et al.,**[14] reported an observational study (239 patients with statins, 114 without statins), that showed statin treatment was not associated with CIN prevention, after adjusting for the propensity of receiving statins (OR= 1.6, 95% CI: 0.86–3.22, P =0.12). In contrast, another study based on a database of 29,409 patients undergoing emergent and non-emergent PCI [15] reported that patients using statins had a lower risk of CIN than did those not using statins (4.4% vs. 5.9%, P,0.001).

In agreement with our results **Xinwei et al.** [16] reported that high-dose simvastatin (80 mg) was more effective than low-dose simvastatin (20 mg) in protecting against renal dysfunction after PCI. Similarly, Also **Quintavalle et al.,** [17] reported that high-dose atorvastatin (80 mg) administered within 24 hours before contrast injection was effective for reducing the incidence of CIN. In contrast to our results in study using simvastatin and atorvastatin failed to demonstrate a beneficial effect on preventing CIN [18].

Our findings demonstrated that the incidence of CIN in rosuvastatin-treated patients was similar to that in atorvastatin- treated patients; the patients were relatively well balanced with respect to their baseline clinical and angiography characteristics. Although we did not demonstrate that rosuvastatin was superior to atorvastatin for preventing CIN, the results may not be surprising considering that different factors are involved in CIN development and that different pathophysiological mechanisms coexist. The present study also demonstrated it was observed that CIN was associated with age > 60 years ,among patient had hypertension or DM , those patient with EF <50 and HB level ≤ 12 , those had dyslipidemia and their baseline Scr> 1 ($p<0.01$) , while gender or CRP were not statistically significant

Two prospective trials showed the protective effects of rosuvastatin on CIN. **Leoncini et al.,** [19]reported that high-dose rosuvastatin (40 mg) administered before PCI prevented CIN in patients with non-ST-segment elevation ACS. **Han et al.,**[20]also reported that a 5-day rosuvastatin treatment significantly reduced the risk of CIN in patients with diabetes and chronic kidney disease undergoing contrast medium injection. Our results confirm and extend these data on the beneficial effect of rosuvastatin.

However, another group **(Quintavalle et al.,)** [17]enrolled 410 patients with CKD in an RCT and demonstrated that a single high dose of atorvastatin administered within a 24 h period before CM exposure, was effective at reducing the CIN rate. Similar findings have been reported from subsequent RCTs [8]. A previous meta analysis of 7 RCTs, with a total of 1399 patients (693 patients receiving high-dose statins, 706 receiving low-dose or no statins) revealed that atorvastatin was beneficial for preventing of CIN [21] which is in agreement with our results**.**

Two large RCTs demonstrated that rosuvastatin pretreatment, upon admission, could reduce CIN occurrence in patients undergoing PCI. **Leoncini et al.,**[19]reported that acute coronary syndrome patients, without ST-segment elevation, who were treated with rosuvastatin (40 mg on-admission, followed by 20 mg/day) experienced less CIN than patients not receiving rosuvastatin. Similarly, in patients with type 2 DM and CKD, another group showed that rosuvastatin significantly reduced the risk of CIN after CM exposure [20].

However, whether the difference (hydrophilic and lipophilic) between statins influences their ability to reduce CIN risk is unclear. Rosuvastatin, a hydrophilic statin, has acute pleiotropic effects, and has been demonstrated to reduce LDL more aggressively, without increasing complications, and improve patient prognosis better than the other statins [22]**;** it also, exerts a beneficial reno-protective effect in patients with renal dysfunction[23]. Additionally, rosuvastatin has a longer plasma half-life and stronger anti-inflammatory effects than atorvastatin [24]**.** Because patients with CKD have significantly higher mean CRP levels [25]rosuvastatin may be more effective in these patients. Furthermore, **Ferreira et al.,** [26] demonstrated that rosuvastatin performed better

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than atorvastatin or simvastatin, in an experimental murine model of cigarette smoke-induced acute lung inflammation, because of better attenuation of both inflammation and oxidative stress parameters. A recent meta-analysis reported that rosuvastatin might also increase apolipoprotein A-I levels at all doses more than atorvastatin [27].

Based on these difference between rosuvastatin and atorvastatin, we hypothesized that rosuvastatin would differ from atorvastatin with respect to their abilities to prevent CIN. Our study reaveled that pretreatment loading dose with Rousvastatin had a similar effect as Atovastatin regarding the development of CIN in patients undergoing PCI.

CONCLUSION

In conclusion, our study found that high dose Rosuvastatin pretreatment exerts an effect similar to atorvastatin in preventing CIN in patients undergoing PCI with. High-dose Rosuvastatin or Atorvastatin loading before PCI was associated with a significantly lower incidence of CIN in patients with ACS.

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