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Clinical Outcomes of Different Atorvastatin Loading Dose before Selective Percutaneous Coronary Intervention

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ABSTRACT:

Background: A high loading dose of atorvastatin 80mg/d has been exposed to be beneficial in stable coronary artery disease and acute coronary syndrome sittings. However, little is known about the long and mid-term clinical outcomes behind the beneficial effects of atorvastatin loading-dose atorvastatin treatment before undergoing percutaneous coronary intervention (PCI). We aim to compare the safety and the clinical outcomes of different atorvastatin loading doses before PCI.

Methods: In this controlled randomized clinical trial, 221 patients who underwent elective PCI, were randomly assigned into three groups to receive either atorvastatin (20 mg; n=84 group A), (40 mg; n=76 group B) or (80 mg; n=83 group C) 12hours before the procedure. The endpoint of this study was the occurrence of major cardiovascular events at 30-day and after 24 months of follow-up.

Results: The occurrence of cumulative MACE at 30-day follow-up was significantly higher in the A group than in the statin group (B group or C group). No cardiac death was noticed in the three groups during in hospital and at 30-days stays in the three groups. At 24 months after follow-up, the occurrence of cumulative MACE was also higher in the A group than in the B or C groups (12.0% in AG vs. 9.3% in BG vs. 5.2% in CG, p = 0.04). No spontaneous MI, cardiac death was noticed in the three groups; the incidence of stroke was 4.3% in group A vs. 0.0% at group B and group C, p = 0.231.

Conclusion： Our study concluded that the administration of atorvastatin as high loading pre-PCI within 24 hours was well accepted and had beneficial anti-inflammatory, and myocardial protective effects in patients with CAD.

Keywords: atorvastatin, percutaneous coronary interventio， homocysteine, neutrophil, highly sensitive C-reactive protein, myocardium protection.

I. INTRODUCTION:

For over 20 years, PCI has been one of the fundamental treatment strategies for either stable CAD or ACS[1]. An injury to the myocardium after PCI procedure commonly caused by procedural complications such as distal embolization, coronary dissection, side-branch occlusion, or disruption of collateral flow. The incidence of myocardial damage post-PCI, which based on the significant elevation of cardiac biomarkers, is 1-30%[2]. During the coronary intervention procedure, mechanical disruption of an atheromatous plaque may occur, which may bring out direct injury to the endothelium the vascular wall, resulting in the activation of the local inflammatory

factors such as C- reactive protein(CRP), interleukin 6 (IL-6), macrophage capping protein (MCP)-1, homocysteine and other inflammatory factors characterized by the adhesion and infiltration of leukocytes at the site of injury[3].

The beginning of the potent statins such as Atorvastatin conducted several trials which suggested a significant reduction of cardiovascular events, especially for those with or without coronary artery disease. Studies, such as the ASCOT-LLA trial was carried out in those patients without CAD. While the MIRACLE and PROVE IT studies were carried out in those patients with CAD[4-7]. Therefore, there is clear evidence that aggressive lipid-lowering has a definite beneficial effect on the primary and secondary prevention of CAD. That is more effective with the most potent statins such as Atorvastatin. Indirect evidence from WOSCOPS, ASCOT, and the CARE trials, in terms of subgroup analysis, has revealed that the relative risk reduction in cardiovascular mortality and morbidity in individuals of the statins group still significant, despite comparable serum cholesterol levels with the placebo group, which brought to the importance of the non-lipid lowering actions of statins in cardiovascular protection[8]. The pleiotropic effects of statins in coronary artery disease are mediated at the level of vascular endothelial cells, platelets, inflammatory cells, and the myocardium itself[8]. Statins are protective when given acutely during any of two phases of myocardial ischemia-reperfusion, the first being before ischemia and the second being at the onset of reperfusion. Experimental models have confirmed that the advantageous acute effects of statins, which can be observed when administered from up to 72 hours before ischemia-reperfusion[9]. High homocysteine (HCY) levels considered as a risk factor for the development and evolution of atherosclerosis. HCY affects endothelial function, causing a prothrombotic environment, platelet activation and endothelial- leukocyte interactions[10]. As well, HCY promotes inflammatory responses that recognized for their role in atherosclerotic disease[11, 12]. Recent studies[13, 14] suggested that inflammatory markers might reflect different aspects of the atherothrombotic process and have a potential role in the prediction of risk for developing coronary artery disease (CAD). Dudman et al[15] study showed that when HCY-induced neutrophils adhered to Human Umbilical Vein Endothelial Cells (HUVECs), the endothelial cells became adequately damaged to leak and detached from their extracellular substrate.

C-reactive protein (CRP) is another pro-inflammatory factor that has been occupied in the pathogenesis of CAD. One of the earliest studies[16] from 1982 showed a correlation between peak CRP concentrations with CK-MB (γ=0.441, $p < 0.001$). Also, those with a complicated myocardial infarction course had an extended increase in CRP. For the duration of the periprocedural period, the increase in serum CRP concentrations follows the increase in serum IL-6 level by 12-36 hours, reaching its peak value by 24 hours after the procedure[17]. Myocardial necrosis is associated with increased inflammatory biomarkers[18]. Elevation of both preprocedural or post-procedural CRP is an independent predictor of a higher incidence of MACE [19, 20]. Cardiac troponins, which included T (cTnT) and I (cTnI), are extremely sensitive and specific cardiac markers to detect myocardial cell injury and necrosis. The predictive value of troponins is now well recognized for patients presenting with acute coronary syndromes (ACS)[21-23] rise of troponin levels after routine PCI has also been known as a prognostic of both short- and long-term major adverse cardiovascular events (MACE)[24].

The objective of this study was to study the changes of the inflammatory factors after PCI and to compare the acute effects of early administration of different loading doses of atorvastatin therapy before PCI on PCI-related inflammatory markers and myocardial injury. And to examine the impact of Atorvastatin loading dose on the incidence of major adverse cardiovascular and cerebrovascular events (MACCE) in patients undergoing elective PCI.

II. MATERIALS AND METHODS:

Patients selection: This is a randomized, prospective clinical trial, which approved by the Ethics Review Boards of our hospital. All patients accepted the agreement for the donation of a sample their blood to be used for scientific purposes. All of the 221 patients suffered from CAD admitted to the Department of Cardiology. Age was between 45 and 72 (58.72±7.82) years old, 165 cases (74.4%) are males. The eligible patients (n=221) were randomly categorized into three groups: group A (20mg/12h before PCI; n=70), group B (40 mg/12h before PCI; n=75) and group C (80 mg/12h before PCI; n=76). Coronary heart disease (CHD): was known as suffering luminal diameter stenosis ≥75% in at least one of 3 major coronary arteries diagnosed by coronary angiography and indicated for PCI. Inclusion criteria: Patients who have been clinically diagnosed with CAD and the coronary angiography result shows stenotic vessel diameter >70%, before interventional therapy, 50-70% stenosis of ischemic lesions with clinical evidence the surgeon decided whether treated with PCI; patients who have no contraindications for statins medications, no extreme adverse reactions after taking statins; normal cardiac enzymes before PCI. Exclusion criteria: acute ST-elevation myocardial infarction (STEMI); acute non ST-elevation myocardial infarction (NSTEMI); requiring urgent PCI; aortic aneurism or dissection; Ejection fraction (EF) <30%; acute cerebrovascular accident; hematologic disorders; current trauma; infectious disease within the last 15 days; severe obstructive pulmonary disease; elevated liver enzymes (aspartate-amino- transferases/ alanine aminotransferases), impaired renal function with a serum creatinine level >133umol/L; history of muscle disorders; history of systemic inflammatory disease or cancer.

Treatment and procedures: All of the selected patients received aspirin 100, clopidogrel 75 mg daily then followed by a loading dose of aspirin (300 mg) and clopidogrel (300mg) administered within 12h before stenting. Standard techniques performed PCI through radial artery puncture. All of the patients received adequate doses of heparin, nitroglycerin, and contrast agent during the PCI procedure. The transradial PCI technique was conducted in patients with coronary lesions that required treatment either with single, double or triple stents technique. After stenting, all of the patients continued the regimen treatment of dual antiplatelet therapy (DAPT), including aspirin (100 mg/day) indefinitely, clopidogrel (75 mg/day) for ≥one year, atorvastatin (20 mg/day), β- blockers and angiotensin- converting enzyme (ACE) inhibitors if there were no contraindications, regardless of the primary randomization assignment.

Statistical analysis: In this study, SPSS 22.0 statistical software (SPSS Inc, Chicago, IL, USA) was applied for all of the analysis processes. Continuous variables are presented as the mean ± standard deviation. We used Kolmogorov–Smirnov test (K–S test or KS test) to estimate the normal distribution of continuous variables, multi-groups were compared by ANOVA, analyzing data was done by using χ^2 test, Correlation analysis was done by linear regression and Pearson correlation coefficient. P-value < 0.05 was considered as statistically significant.

III. RESULTS:

General Characteristics: Clinical features in the three groups are reported in Table 1. All the groups had the same baseline properties, including age, gender, body mass index (BMI), clinical manifestations, EF, and medication used during hospitalization (all P>0.05). Table 1.

Table 1 Main clinical features of patients in the three groups

Variable	Group A(20mg) n=70	GroupB(40mg) n=75	GroupC(80mg) n=76	P
Age(y)	58.47±8.25	58.77±8.00	58.72±7.34	0.943
Gender (male%)	51(72)	55 (73)	59 (77)	0.745
BMI, kg/m2	23.25±4.27	23.87±3.87	22.70±3.82	0.197
Diabetes mellitus	60(14)	66(12)	60(21)	0.312
Hypertension	30 (42)	38 (50)	34 (44)	0.839
Smokers	41 (58)	35 (46)	43 (44)	0.099
Previous PCI	0(0)	0(0)	0(0)	1
Previous CABAG	0(0)	0(0)	0(0)	1
LVEF, %	62±6	59±7	60±7	0.69
RBS, mmol/L	5.82±0.97	6.05±1.53	6.17±1.63	0.321
LDL-C, mmol/L	2.96±0.94	2.64±1.16	2.40±1.09	0.212
TG, mmol/L	3.35±1.51	3.28±1.60	3.54±1.54	0.579
Cholesterol, mmol/L	5.42±1.52	5.35±2.35	5.13±1.40	0.587
HDL-C, mmol/L	1.30±0.42	1.15±0.42	1.28±0.44	0.75
Variable	Group A(20m g) n=70	GroupB(40mg) n=75	GroupC(80mg) n=76	P
Creatinine, mg/L	71.16±19.63	71.93±19.70	73±23.15	0.865
AST, IU/L	37.19±19.74	42.07±33.21	41.10±25.14	0.513
ALT, IU/L	41.54±23.02	37.91±23.66	43.88±25.05	0.306
Aspirin	100%	100%	100%	1
Clopidogrel	100%	100%	100%	1
Beta-blockers	74.2%	77.6%	90.8%	0.432
ACEI/ARB	30.2%	28.4%	38.6%	0.312

Note: data shown in this table are presented as n (%) or mean ± SD. HDL-C High-density lipoprotein cholesterol; LDL-C Low-density lipoprotein cholesterol; TG Triglycerides; PCI percutaneous coronary intervention; CABG Coronary Artery Bypass Grafting; LVEF Left ventricular ejection fraction; RBS Blood Sugar HCY homocysteine; hs-CRP highly sensitive C-reactive Protein; ALT Alanine Aminotransferase; AST Aspartate Aminotransferase; ACEI/ARB Angiotensin-converting enzyme inhibitor or angiotensin receptor blocker.

Coronary interventional Parameters: Procedural features in all groups are presented in Table 2. All patients were successfully treated by implantation of drug-eluting stents. There were no significant differences in the basic coronary-angiographic parameters. (all P>0.05). Table 2

Comparison of involved coronary artery lesions and stents number among the three groups: A total of 221 patients had successfully undergone PCI operation, no patients suffered coronary artery dissection, coronary artery rupture, acute or subacute thrombosis, heart failure, death or another major adverse cardiac event. According to the study results of coronary angiography and stenting statistical analysis, no differences were found in the Multivessel lesions and implanted stents numbers for all groups (P>0.05). Table 2.

Table 2 Coronary-interventional parameters in the three groups

Variable	A(20mg) n=70	B(40mg) n=75	C(80mg) n=76	P
Vessels treated				
Left main artery	7(10)	8 (11)	10(13)	0.817
LAD	51(72)	48(64)	46(60)	0.143
LCX	16(23)	31(41)	31(41)	0.061
RCA	30 (43)	45(60)	36 (47)	0.081
Involved vessel lesions	46(66)	54(72)	64(84)	0.094
Single vessel	24(34)	21(28)	12(16)	0.102
Douple vessel	43(61)	50(66)	61(80)	0.066
Triple vessel	3(5)	4(6)	3(4)	0.258
Number of stents	1.69±0.81	1.95±0.77	1.70±0.71	0.065
Duration of procedures	41.39±14.80	44.77±18.011	42.33±17.24	0.453
GpIIb/IIIa inhibitor	61(87)	64(85)	66(87)	0.943

Data presented as n (%) or mean ± SD.LAD left descending anterior coronary artery; LCX left circumflex artery; RCA right coronary artery; min minute; GpIIb/IIIa inhibitor Glycoprotein IIb/IIIa inhibitor.

Procedural clinical Outcomes

The occurrence of cumulative MACE at 30-day follow-up was significantly higher in the A group than in the statin group (B group or C group) (5.7% in group A and 1.3% in group B vs. 0.0% in group C, p = 0.032). The incidence of periprocedural MI (4.5% in A group vs 2.7% in B group vs 1.3.0% in C group, p = 0.0001). The occurrence rates of cardiac death, and spontaneous MI, were similar in the three groups; the occurrence of rehospitalization was higher in the A group than in groups B and C, which was not statistically significant, (7.1%, 2.6%, and 2.6% in the three groups, respectively, p = 0.293) (Table 3).

Table 3: The 30-days and in-hospital post PCI outcomes

Variable	A(20mg) n=70	B(40mg) n=75	C(80mg) n=76	P
Cumulative MACE % (pts)	4 (5.7%)	1 (1.3%)	0(0.0%)	0.032
Cardiac death % (pts)	0 (0.0%)	0 (0.0%)	0 (0.0)	1
Periprocedural MI % (pts)	4 (5.7%)	2 (2.7%)	1 (1.3%)	0.305
Spontaneous MI % (pts)	0 (0.0%)	0 (0.0%)	0 (0.0)	1
Rehospitalization % (pts)	5 (7.1%)	2 (2.6%)	2 (2.6%)	0.293

At 24 months after follow-up, the occurrence of cumulative MACE was also higher in the A group than in the B or C groups (12.0% in AG vs. 9.3% in BG vs. 5.2% in CG, p = 0.04). Furthermore, the rate of the spontaneous MI, cardiac death was 0 the three groups, respectively; the incidence of stroke was 4.3% in the AG vs. 0.0% at BG and CG, p = 0.231; the prevalence of rehospitalization was 3.4% in the A vs. 1.8% at group B vs. 1.3% at group C, p = 0.293. For more details see Table 4.

Table 4: The 24-months post PCI clinical outcomes

Variable	A(20mg) n=70	B(40mg) n=75	C(80mg) n=76	P
Cumulative MACE % (pts)	8 (12%)	7 (9.3%)	4(5.2%)	0.04
Cardiac death % (pts)	0 (0.0%)	0 (0.0%)	0 (0.0)	1
Periprocedural MI % (pts)	4 (5.7%)	2 (2.7%)	1 (1.3%)	0.305
Spontaneous MI % (pts)	0 (0.0%)	0 (0.0%)	0 (0.0)	1
Stroke % (pts)	3 (4.3%)	0 (0.0%)	0 (0.0)	0.231
Rehospitalization % (pts)	2 (3.4%)	1 (1.8%)	1 (1.3%)	0.293

IV. DISCUSSIONS:

Results of the current study advise that a single reloading dose of atorvastatin (80, 40, or 20 mg) within 24 hours before elective PCI can effectively reduce the incidence of peri-procedural MI in CAD patients.

There is a clear clue that aggressive lipid reduction is of definite advantage in primary and secondary prevention of CAD and that this is the most effective with more strong statins such as Atorvastatin. However, it is now becoming increasingly evident that all the beneficial effects of statins may not be related to their ability to lower cholesterol. Indirect evidence from WOSCOPS, ASCOT, and the CARE trials, in terms of subgroup analysis, has revealed that the relative risk reduction in cardiovascular mortality and morbidity in individuals in the statins group was still significant despite comparable serum cholesterol levels with the placebo group. This brought to the front position of the importance of the non-lipid lowering actions of statins in cardiovascular protection[8].

Experimental models have confirmed that the advantageous acute effects of statin, which can be observed when administered from up to 72 hours before ischemia-reperfusion until just before ischemia or reperfusion[9]. To the best of our knowledge, the current study is one of the numerous studies to demonstrate that atorvastatin loading in patients with CAD undergoing primary PCI could prevent post PCI major cardiovascular events (MACE). This may be attributed to the effects of the Atorvastatin loading dose on the myocardial perfusion improvement. This study designed to compare the effects of different loading doses administered orally of 20mg, 40mg, and 80mg atorvastatin 12 hours before PCI procedure, in patients with CAD, on post PCI major cardiovascular events. Many studies showed that the applying of concentrated statins drugs before coronary intervention therapy could improve myocardial perfusion, diminished significant adverse cardiac events(MACEs), and have been demonstrated to be relatively safe[25-28]. Atorvastatin for Reduction of Myocardial Damage during Angioplasty (ARMYDA) trial was the first investigation of patients who never used statins before PCI, atorvastatin dose of 40mg/day was administrated for seven days prior PCI in patients with stable coronary disease[29]. In ARMYDA-Acute Coronary Syndromes (ACS)[30] and ARMYDA -REC APTURE[25], atorvastatin dose schedule was 80mg 12 h pre-PCI and an additional 40 mg was given just before PCI procedure. In the Novel Approaches for Preventing or Limiting Events (NAPLES) II trial[31], a loading dose of 80mg atorvastatin was administered the day before elective PCI. In both of these four trials, atorvastatin has been shown a significant reduction in the levels of markers of myocardial injury compared with placebo post-PCI. The endpoint was the incidence of perioperative MI (which defined as higher than the upper limit of normal CK-MB 2 times); MI patients in the experimental group rate was reduced from 18% to 5% ($P = 0.05$)[30].

A study has shown 30-days after PCI, the incidence of myocardial infarction, target vessel revascularization, cardiac death, and the percent of the MACE rates was decreased from 17% to 5%, and this benefit of perioperative myocardial infarction rate was significantly reduced (5% VS 15%, $P = 0.04$).

V. CONCLUSIONS

Our study discovered that pre-PCI loading with statins might reduce the degree of PCI-induced myocardial injury; also, these beneficial acute effects of statins on the myocardium increased with increasing dose of statin loading before PCI. This loading dose treatment might be related to mild adverse reactions and be well tolerated. The present study proposes that different loading protocols before PCI procedure may be used clinically relying on the individual conditions of the patient.

Limitations

Our study has limitations. This study is a small sample single-center observational study and a small sample of cases, so some necessary information between groups is not balanced enough. In this study, the time taking of atorvastatin pre-treatment in each group was not similar, did not separate stable angina patients from unstable angina patients, so this study has certain limitations.

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