Comparative Study of Low Doses of Rosuvastatin and Atorvastatin on Lipid and Glycemic Control in Patients with Metabolic Syndrome and Hypercholesterolemia

Jong-Seon Park[1], Young-Jo Kim[1], Ji-Yong Choi[2], Yoon-Nyun Kim[3], Teck-Jong Hong[4], Dong-Soo Kim[5], Ki-Young Kim[6], Myung-Ho Jeong[7], Jei-Keon Chae[8], Seok-Kyu Oh[9], and In-Whan Seong[10]

Departments of Internal Medicine, [1]Yeungnam University Medical Cencer, Daegu; [2]Daegu Catholic University Medical Center, Daegu; [3]Keimyung University Dongsan Medical Center, Daegu; [4]Pusan National University Hospital, Busan; [5]Inje University Pusan Paik Hospital, Busan; [6]Konyang University Hospital, Daejeon; [7]Chonnam National University Hospital, Gwangju; [8]Chonbuk National University Hospital, Jeonju; [9]Wonkwang University Hospital, Iksan; [10]Chungnam National University Hospital, Daejeon, Korea

Background/Aims: This multicenter, open-labeled, randomized trial was performed to compare the effects of rosuvastatin 10 mg and atorvastatin 10 mg on lipid and glycemic control in Korean patients with nondiabetic metabolic syndrome.

Methods: In total, 351 patients who met the modified National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria for metabolic syndrome with low-density lipoprotein cholesterol (LDL-C) levels ≥ 130 mg/dL were randomized to receive either rosuvastatin 10 mg (n = 173) or atorvastatin 10 mg (n = 178) for over 6 weeks.

Results: After 6 weeks of treatment, greater reductions in total cholesterol (- 35.94 ± 11.38 vs. - 30.07 ± 10.46%, $p < 0.001$), LDL-C (48.04 ± 14.45 vs. 39.52 ± 14.42%, $p < 0.001$), non-high-density lipoprotein cholesterol (- 42.93 ± 13.15 vs. - 35.52 ± 11.76%, $p < 0.001$), and apolipoprotein-B (- 38.7 ± 18.85 vs. - 32.57 ± 17.56%, $p = 0.002$) levels were observed in the rosuvastatin group as compared to the atorvastatin group. Overall, the percentage of patients attaining the NCEP ATP III goal was higher with rosuvastatin as compared to atorvastatin (87.64 vs. 69.88%, $p < 0.001$). Changes in glucose and insulin levels, and homeostasis model assessment of insulin resistance index were not significantly different between the two groups. The safety and tolerability of the two agents were similar.

Conclusions: Rosuvastatin 10 mg was more effective than atorvastatin 10 mg in achieving NCEP ATP III LDL-C goals in patients with nondiabetic metabolic syndrome, especially in those with lower NCEP ATP III target level goals. **(Korean J Intern Med 2010;25:27-35)**

Keywords: Metabolic syndrome X; Hypercholesterolemia; Rosuvastatin; Atorvastatin

INTRODUCTION

Metabolic syndrome consists of a group of cardiovascular risk factors, namely dyslipidemia, high blood pressure (BP), abdominal obesity, and insulin intolerance, whose concurrent appearance increases the risk of atherosclerotic cardiovascular disease [1]. Using the modified National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III [ATP III]) criteria, the prevalence of atherosclerotic cardiovascular disease is estimated to be as high as 24.8% in Korea and is
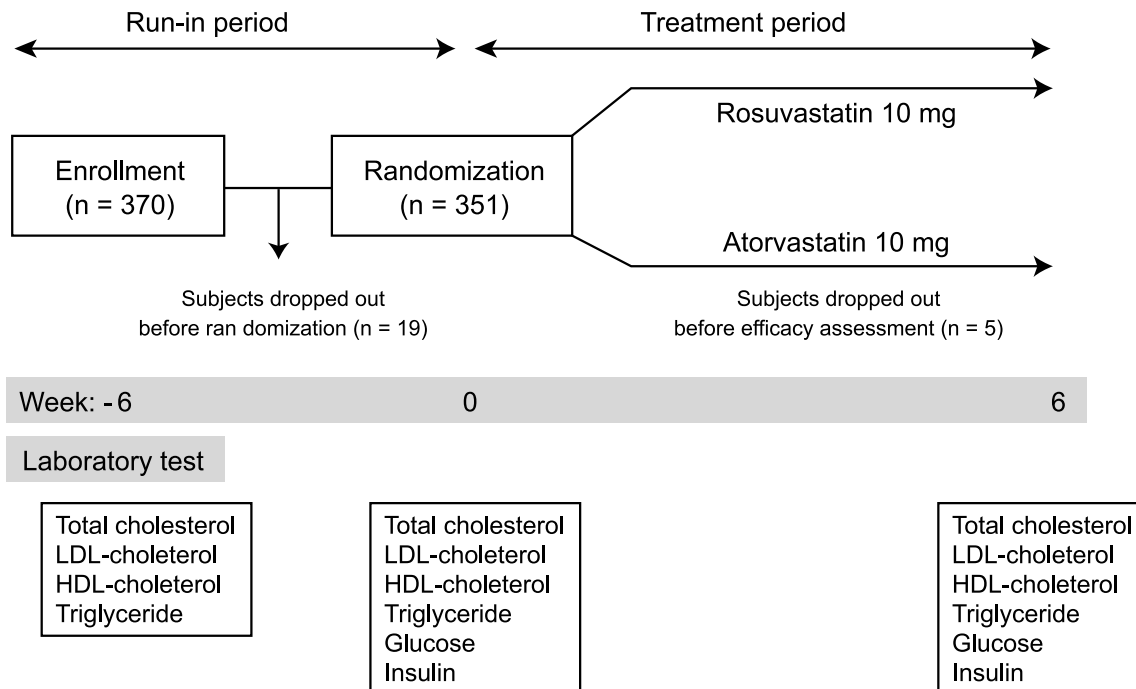
Figure 1. Trial design. LDL, low-density lipoprotein; HDL, high-density lipoprotein.

continuing to rapidly increase to epidemic proportions [2]. Elevated cholesterol levels have also been shown to be a strong risk factor for the development of coronary heart disease (CHD). This clustering of risk factors may interact synergistically to affect atherosclerosis and cardiovascular events [3]. Current guidelines for lipid management stress the importance of low-density lipoprotein cholesterol (LDL-C) levels as the primary goal of therapy [4]; however, a high proportion of patients, especially those having high lipid levels, do not achieve their target LDL-C levels despite lipid-lowering therapy [5,6].

Statins effectively lower blood cholesterol levels and reduce the risk of cardiovascular events in many patient types, and are therefore recommended as first-line agents for lowering LDL-C levels [4,7]. Statins also improve other aspects of the lipid profile, such as increasing high-density lipoprotein cholesterol (HDL-C) and lowering triglyceride levels to some extent.

Rosuvastatin is a highly effective HMG-CoA reductase inhibitor, which was registered in 2002 in Korea. Rosuvastatin use has been previously shown in numerous studies to be associated with greater LDL-C level reductions as compared to atorvastatin, simvastatin, or pravastatin use [8-10]. The primary objective of the current trial was to compare the effects of rosuvastatin 10 mg with that of atorvastatin 10 mg, which are the lowest-dose tablets available, on the percentage of patients who reach the NCEP ATP III LDL-C goal and safety in subjects with nondiabetic metabolic syndrome after 6 weeks of treatment. The secondary objective was to compare the effects of rosuvastatin with that of atorvastatin on glucose control and insulin resistance.

METHODS

Study design

This 6-week, multicenter, randomized, open-label, parallel-group, single-dose trial (NCT00335699) was designed to compare the efficacy of a single dose of rosuvastatin and atorvastatin in patients having nondiabetic metabolic syndrome with dyslipidemia (Fig. 1). The study was conducted from August 2005 to January 2006 at 20 medical centers in Korea. The study included a 6-week dietary run-in period before randomization, followed by a 6-week treatment phase. Subjects entering the run-in period were asked to follow the NCEP Step I diet and required to discontinue any previous lipid lowering therapy. Following the dietary lead-in period, patients with fasting LDL-C levels ≥ 130 mg/dL to < 220 mg/dL were selected

Table 1. Baseline characteristics at randomization

Factors	Rosuvastatin 10 mg (n = 172)	Atorvastatin 10 mg (n = 178)	p value
Sex, male, %	73 (42.44)	67 (37.64)	0.359
Age, yr	60.49 ± 0.74	58.96 ± 0.75	0.148
Weight, kg	66.84 ± 0.81	66.73 ± 0.83	0.924
Height, cm	160.01 ± 0.75	158.80 ± 0.64	0.221
Systolic BP, mmHg	134.58 ± 1.15	135.73 ± 1.14	0.477
Diastolic BP, mmHg	80.16 ± 0.78	80.18 ± 0.82	0.988
Waist circumference, cm	91.81 ± 0.47	91.60 ± 0.54	0.760

Values are presented as the mean ± SE.
BP, blood pressure.

Table 2. Changes in metabolic parameters after treatment

	Rosuvastatin 10 mg (n = 170)		Atorvastatin 10 mg (n=176)		p value[c]
	Baseline	Treatment	Baseline	Treatment	
Lipids, mg/dL					
Total cholesterol	237.52 ± 2.07	151.70 ± 2.26[b]	238.55 ± 1.89	166.23 ± 2.01[b]	< 0.0001
LDL-C	163.64 ± 1.76	84.37 ± 1.81[b]	163.85 ± 1.62	98.37 ± 1.76[b]	< 0.0001
HDL-C	39.66 ± 0.54	39.15 ± 0.55	39.76 ± 0.55	38.55 ± 0.57[a]	0.448
Triglyceride	171.08 ± 5.20	140.92 ± 4.64[b]	174.75 ± 4.69	146.56 ± 4.76[b]	0.397
Non-HDL-C	197.85 ± 1.90	112.55 ± 2.12[b]	198.80 ± 1.77	127.69 ± 1.88[b]	< 0.0001
Apolipoprotein A-1	141.95 ± 2.01	141.65 ± 1.76	141.75 ± 1.92	140.71 ± 2.43	0.756
Apolipoprotein B	117.44 ± 1.55	71.12 ± 1.69[b]	117.90 ± 1.38	78.62 ± 1.50[b]	0.001
Glucose and insulin					
Glucose, mg/dL	91.08 ± 0.75	91.07 ± 0.95	93.32 ± 0.84	91.86 ± 0.88	0.541
HbA1c, %	5.88 ± 0.04	5.93 ± 0.04[a]	5.86 ± 0.03	5.86 ± 0.03	0.456
HOMA index	1.21 ± 0.07	1.57 ± 0.11[a]	1.52 ± 0.18	1.45 ± 0.11	0.465
hsCRP, mg/L	2.29 ± 0.30	2.24 ± 0.42[a]	2.23 ± 0.35	1.77 ± 0.26[a]	0.344

Values are presented as the mean ± SE.
LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; HbA1c, hemoglobin A1C; HOMA, homeostasis model assessment; hsCRP, high-sensitivity C-reactive protein.
[a] $p < 0.05$ vs. baseline.
[b] $p < 0.001$ vs. baseline.
[c] Statistical difference between rosuvastatin and atorvastatin after treatment.

and randomly assigned to two parallel treatment groups. At baseline, eligible subjects were randomized 1 : 1 to receive either rosuvastatin (Astra-Zeneca Korea, Seoul, Korea) 10 mg or atorvastatin (Pfizer Pharmaceuticals Korea, Seoul, Korea) 10 mg once daily at bedtime for 6 weeks. The study drug was discontinued and subjects were removed from the study if they withdrew informed consent, became pregnant, or developed creatine kinase levels greater than 10 times the upper normal limit.

The ethics committees and institutional review boards at each participating hospital approved the study protocol. All patients provided informed consent to participate in this study.

Subjects

Patients were ≥ 18 years of age and had nondiabetic metabolic syndrome. Metabolic syndrome was defined according to the modified NCEP ATP III criteria [11], which requires at least three of the following: abdominal obesity (waist circumference): men > 90 cm (36 inches), women > 80 cm (32 inches); triglyceride levels ≥ 150 mg/dL (1.70 mmol/L); HDL-C levels: men < 40 mg/dL (1.04 mmol/L) and women < 50 mg/dL (1.3 mmol/L); BP ≥ 130 / ≥ 85 mmHg or subject receiving antihypertensive
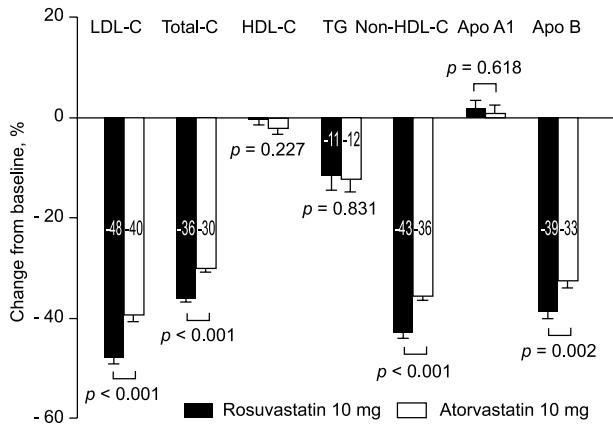
Figure 2. Percent change of lipid and apolipoprotein after treatment for 6 weeks.
LDL-C, low-density lipoprotein cholesterol; Total-C, total cholesterol; HDL-C, high-density lipoprotein cholesterol; Apo, apolipoprotein.

Figure 3. Proportions of patients reaching different Adult Treatment Panel III, low-density lipoprotein cholesterol (LDL-C) level goals at 6 weeks after treatment.
[a] Proportion of patients reaching LDL-C level goal < 100, 130 or 160 mg/dL depending on risk category at baseline.

treatment; and fasting blood glucose 110 mg/dL (6.11 mmol/L) to 125 mg/dL (6.94 mmol/L). Patients were excluded if they were pregnant or had malignancy. Additional exclusion criteria included diabetes, and active arterial disease such as unstable angina, myocardial infarction, cerebrovascular accident, coronary artery bypass surgery, or angioplasty within 2 months prior to enrollment. After completing the 6-week dietary run-in period, fasting LDL-C concentrations were required to be ≥ 130 mg/dL (3.36 mmol/L) to < 220 mg/dL (5.69 mmol/L) and fasting triglyceride levels were required to be < 400 mg/dL (4.52 mmol/L).

Assessments

Sample analysis for efficacy endpoints was performed in the Green Cross Reference Laboratory, Yongin, Korea, which was certified by the American College of Pathology (LAP No. 6708401) and the National Committee for Clinical Laboratory Standards. Blood samples from patients who had fasted for 12 hours were collected at all investigational sites and delivered by courier to the central laboratory within 24 hours of blood draw. To assess the primary efficacy endpoint, lipid parameters such as total cholesterol, LDL-C, HDL-C, and triglyceride levels were measured during the dietary lead-in period, at randomization, and 6 weeks after treatment. Additionally, levels of apolipoprotein A-1 and B, high-sensitivity C-reactive protein (hsCRP), insulin, glucose, and hemoglobin A1c (HbA1c) were measured at randomization and at 6 weeks after treatment. LDL-C levels were calculated using the
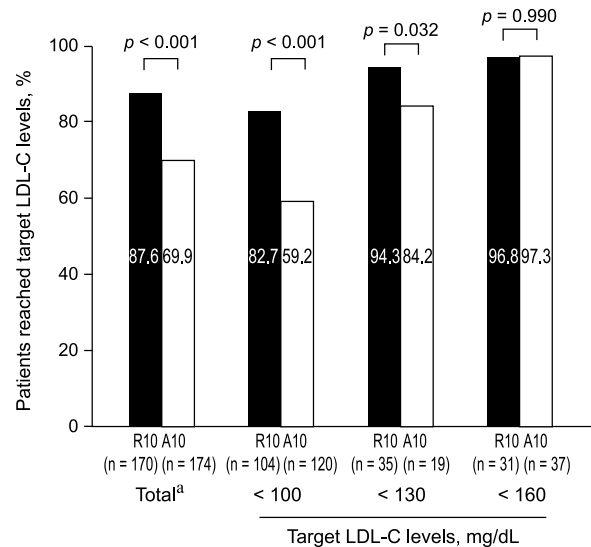
Friedewald equation (LDL-C = total cholesterol - (HDL-C + triglyceride/5). The insulin resistance index was estimated using the homeostasis model assessment (HOMA) for insulin resistance based on the following formula: fasting serum insulin (μU/mL) × fasting plasma glucose (mmol/L)/22.5. According to the NCEP ATP III guidelines, the goal LDL-C level for each patient and the proportion of patients achieving the goal in each group was assessed. Persons with CHD or CHD risk equivalent (Framingham 10-year CHD risk > 20%) had a LDL-C level goal of < 100 mg/dL. Those with multiple risk factors had a LDL cholesterol level goal of < 130 mg/dL and those with 0 - 1 risk factor (s) had a goal LDL cholesterol of < 160 mg/dL.

Individual demographic data, physical findings, vital signs, and adverse events were evaluated and recorded in the given case record form. To evaluate adverse events, various laboratory assessments including blood counts, and hemoglobin, aspartate aminotransferase, alanine aminotransferase, creatine kinase, electrolyte, and creatinine levels were performed at each time point.

Statistical analysis

One-hundred and forty-three evaluable subjects per treatment group were required to achieve 95% power for detecting a clinically significant difference of 6% at the 5% two-sided level in percentage change from baseline in

Table 3. Percent change of glucose and insulin resistance after treatment for 6 weeks

Factors	Rosuvastatin (n = 170)	Atorvastatin (n = 176)	p value
Glucose	0.24 ± 0.84	- 1.12 ± 0.76	0.231
Insulin	68.14 ± 13.28	67.91 ± 18.20	0.992
HbA1c	1.08 ± 0.53	- 0.10 ± 0.28	0.048
HOMA index	69.79 ± 13.49	72.60 ± 20.84	0.910

Values are presented as the mean ± SE.
HbA1c, hemoglobin A1c; HOMA, homeostasis model assessment.

LDL-C levels at 6 weeks with an assumed standard deviation of 14% [12]. Assuming a dropout rate of 20% during the randomized treatment period, approximately 180 subjects were recruited to each active treatment group. To obtain the required number of randomized subjects (360 in total), approximately 900 subjects were assumed to be needed for screening based on a screening failure rate of 60%.

The primary analysis population was the last observation carried forward on the intention-to-treat population. This included all subjects with a baseline and at least one post-baseline lipid level measurement. All numeric variables were expressed as the mean ± SEM (standard error of the mean). Efficacy endpoints were analyzed using the unpaired *t*-test for continuous variables and Pearson's chi-square test for frequencies with 95% confidence intervals. Multivariate logistic regression analysis was used to evaluate the predictors for reaching target NCEP ATP III LDL-C levels after treatment. Variables used for analysis included the statin used, presence of coronary artery disease and hypertension, body mass index, gender, age, waist circumference, and lipid parameters.

On the basis of the actual treatment received, safety data were evaluated for all patients who received at least one dose of study medication.

RESULTS

Subject characteristics

In total, 645 subjects were screened for participation in this study. Of them, 370 patients entered the dietary lead-in phase and 351 patients met the inclusion criteria and were randomly assigned to treatment with either rosuvastatin 10 mg or atorvastatin 10 mg (Fig. 1). One patient was lost to follow-up and had no safety assessment. Table 1 shows demographic data and baseline characteristics

of all 350 subjects who took at least one dose of the study drug at baseline. In terms of demographic data and baseline characteristics, no statistically significant differences existed between the two treatment groups. Patients had a mean age of 60 years in the rosuvastatin group and 58 years in the atorvastatin group. Mean body weights were 66 kg in the rosuvastatin group and 66 kg in the atorvastatin group. Mean systolic and diastolic BP and waist circumstance were comparable between the two groups (Table 1).

A total of five patients dropped out before efficacy assessment. Data from 346 patients were analyzed for efficacy in the intention-to-treat population defined as those who took at least one dose of study drug and had lipid levels checked at baseline and follow-up. Safety assessments were performed in 350 patients who were randomized and available for follow-up.

Changes in metabolic parameters

Lipid levels, glucose levels, insulin resistance indices, and hsCRP levels at baseline and 6 weeks are shown in Table 2. In each group, atherogenic lipid parameters including total cholesterol, LDL-C, triglyceride, non-HDL-C, and apolipoprotein B had significantly decreased after 6 weeks of treatment ($p < 0.001$ vs. baseline). Only the atorvastatin treatment produced a modest decrease in HDL-C. Rosuvastatin treatment significantly increased HbA1c and the HOMA index; however, no significant change occurred in the atorvastatin group.

Data from two groups were analyzed for an efficacy comparison in the intention-to-treat population. Baseline values of all parameters were similar between the two groups. At 6 weeks after treatment, rosuvastatin 10 mg produced a significantly greater reduction in total cholesterol, LDL-C, non-HDL-C, and apolipoprotein B levels. Otherwise, no significant differences were detected in HDL-C and apolipoprotein A-1 levels between the two

Table 4. Most common treatment-related adverse events (≥ 1% in any treatment group) during the treatment period

Adverse events	Rosuvastatin (n = 172)	Atorvastatin (n = 178)
Any adverse events	13 (7.56)	9 (5.06)
Serious adverse events	1 (0.58)	2 (1.12)
Drug related adverse events	0 (0.00)	5 (2.81)
Adverse events which caused discontinuation of the study	0 (0.00)	1 (0.56)

Values are presented as number (%).

groups. In addition, no significant differences were observed with respect to glucose, HbA1c, and hsCRP levels, and HOMA index between the rosuvastatin and atorvastatin groups at 6 weeks (Table 2). At 6 weeks, LDL-C absolute values decreased by 48.04 ± 14.45 mg/dL in the rosuvastatin group and by 39.52 ± 14.42 mg/dL in the atorvastatin group; the former reduction associated with rosuvastatin use was significantly larger than that with atorvastatin ($p < 0.0001$).

Percent changes from baseline in lipid profiles after treatment for 6 weeks, including LDL-C, are shown in Fig. 2. Reductions in total cholesterol (- 35.94 ± 11.38 vs. - 30.07 ± 10.46%, $p < 0.0001$), non-HDL-C (- 42.93 ± 13.15 vs. - 35.52 ± 11.76%, $p < 0.0001$), and apolipoprotein B (- 38.7 ± 18.85 vs. - 32.57 ± 17.56%, $p = 0.0019$) levels were larger in the rosuvastatin group as compared to the atorvastatin group (Fig. 2).

LDL-C target achievement

According to the reported CHD or/and CHD risk equivalents and/or number of risk factors and/or Framingham 10-year risk, the NCEP ATP III LDL-C target goal was determined in each patient and the success rate in reaching their target goal was analyzed after 6 weeks in each group.

The percentage of patients who reached their ATP III LDL-C level goals was higher in the rosuvastatin group (87.6 vs. 69.9%, $p < 0.001$). Among them, patients having LDL-C target cholesterol level goals of < 100 mg and < 130 mg reached their LDL-C target level goals more frequently in the rosuvastatin group as compared to the atorvastatin group. In contrast, in patients with a LDL-C target level goal < 160 mg, more than 96% reached their target goal without a significant difference between the rosuvastatin- and atorvastatin-treated groups (Fig. 3). The overall achievement rate for NCEP non-HDL-C level target goals after 6 weeks of treatment was 76.08% in the rosuvastatin group and 58.92% in the atorvastatin group ($p = 0.067$).

Percent changes in glucose levels and insulin resistance

Percent changes in glucose levels and insulin resistance at 6 weeks are summarized in Table 3. Changes in glucose and insulin levels were not significantly different between the two groups; however, HbA1c levels were slightly higher in the rosuvastatin group with marginal significance. To evaluate insulin resistance in the two groups, the HOMA index was calculated. At 6 weeks, the HOMA index increased in both groups and the difference between groups was not significant.

Safety

Both rosuvastatin 10 mg and atorvastatin 10 mg were

Table 5. Adjusted odds ratios for LDL-cholesterol goal achievement after treatment for 6 weeks

Factors	Odds ratio (95% confidence interval)	Standard error	p value
Age, yr	0.997 (0.966 - 1.029)	0.016	0.850
Sex, male	0.424 (0.214 - 0.842)	0.350	0.014
Hypertension	1.025 (0.434 - 2.421)	0.439	0.955
Coronary artery disease	3.806 (1.959 - 7.391)	0.339	< 0.001
Body mass index ≥ 25	0.669 (0.353 - 1.267)	0.326	0.217
Waist circumference > 90 cm	1.445 (0.703 - 2.968)	0.368	0.317
Total cholesterol ≥ 230 mg/dL	0.885 (0.390 - 2.006)	0.418	0.769
LDL-cholesterol ≥ 160 mg/dL	0.446 (0.208 - 0.960)	0.391	0.039
Treatment, rosuvastatin	3.26 (1.800 - 5.906)	0.303	< 0.001

LDL, low-density lipoprotein.

well tolerated, with similar incidences of adverse events. During the treatment period, 13 subjects in the rosuvastatin group and 9 subjects in the atorvastatin group reported adverse events (Table 4). The most frequent adverse events in the rosuvastatin group were edema and dizziness, both with incidences of 1.16%. Only five adverse events were reported in the atorvastatin group as related to the study drug; myalgia was reported in one case (0.56%). All adverse events were mild, developed within 2 weeks after starting treatment, had no action taken, and resolved spontaneously. No drug-related adverse effects were observed in the rosuvastatin group. Also, no patient had an increase in alanine aminotransferase level > 3 times the upper limit of normal or rhabdomyolysis.

Predictors for LDL-C level goal achievement at 6 weeks

Overall, the percentage of patients who reached NCEP ATP III LDL-C target level goals was higher in the rosuvastatin group as compared to the atorvastatin group. Univariate analysis showed that patients with target LDL-C levels at 6 weeks tended to be rosuvastatin-treated and have coronary artery disease. Multivariate logistic regression analyses that included age, gender, statin, coronary artery disease, hypertension, body mass index, waist circumference, baseline total cholesterol levels, and triglyceride levels showed that rosuvastatin treatment, the presence of coronary artery disease, female gender, lower total cholesterol level, and lower LDL-C levels at baseline were independent predictors for achievement of target LDL-C levels at 6 weeks (Table 5).

DISCUSSION

This study evaluated the comparative efficacy of the lowest doses available for two effective statins, rosuvastatin and atorvastatin, in Korean patients with nondiabetic metabolic syndrome.

Rosuvastatin 10 mg was more effective than atorvastatin 10 mg in reducing LDL-C levels in subjects with nondiabetic metabolic syndrome after 6 weeks of treatment. Consistent with the greater reductions in LDL-C levels, more patients in the rosuvastatin group achieved LDL-C level goals as compared to the atorvastatin group. Otherwise, no significant difference was observed in glucose levels and insulin resistance.

Metabolic syndrome, especially in the presence of high

LDL-C levels, is already known to increase the risk of cardiovascular mortality and morbidity [13]. Statins are effective in decreasing LDL-C levels in patients with dyslipidemia. Survey studies have demonstrated that in real-world settings, only 67% of patients with treated dyslipidemia reach their LDL-C target level goals [14]. In this study, rosuvastatin treatment was associated with reaching recommended LDL-C level goals in a higher percentage of patients overall as compared to atorvastatin (87.6 vs. 69.9%). In particular, rosuvastatin was more effective in patients requiring more intensive LDL-C level lowering to less than 100 or 130 mg/dL. In high-risk patients with stronger targets of LDL-C levels < 100 mg/dL, rosuvastatin brought 83% of patients in this trial to the ATP III LDL-C level goal, which was higher than achieved in other studies conducted in South-Asian (76%) and Hispanic-American (61%) patients [15,16]. Both statins, however, were effective in patients with high target LDL-C level goals < 160 mg/dL. These data highlight the importance of using highly effective statins in high-risk patients to enable them to achieve their lower NCEP ATP III LDL-C level goals.

With respect to other elements of the lipid profile, improvements in total cholesterol, apolipoprotein B, and non-HDL-C levels were also significantly greater with rosuvastatin as compared to atorvastatin, whereas changes in HDL-C, triglyceride, and apolipoprotein A1 levels were similar in both treatment groups. Unlike other studies in which rosuvastatin effectively raised HDL-C levels [9,15], HDL-C levels in this study were not effectively improved in either group [9].

Metabolic syndrome is associated with an increased risk of both insulin resistance and diabetes [17]. Additionally, changes in the insulin resistance index were investigated by evaluating the HOMA index, which is a positive predictor of metabolic syndrome [18]. Studies in an animal model of insulin resistance suggest that rosuvastatin treatment increases whole-body and peripheral tissue insulin sensitivity via improved cellular insulin signal transduction [19]. In contrast, in our study conducted in nondiabetic subjects, a tendency was detected for an increased HOMA index in both treatment arms. Major changes in this parameter were attributable to high increases in insulin concentrations. The degrees of percent change in fasting glucose, insulin concentrations, and HOMA index were not significantly different between the rosuvastatin and atorvastatin treatment groups. Thus, further studies are needed to elucidate the effects of statins on glucose

metabolism, insulin secretion, and insulin sensitivity under diabetic or nondiabetic conditions.

A multivariate analysis was performed to determine independent predictors of LDL-C goal achievement at 6 weeks. Overall, sex, the presence of coronary artery disease, LDL-C levels, and rosuvastatin treatment were predictive of target LDL-C achievement. Among these factors, rosuvastatin was the strongest predictor, with an odds ratio of 3.26. Moreover, the presence of coronary artery disease was an independent predictor of achieving target LDL-C levels. These patients were assumed to have been more likely to take interest in diet control or exercise than patients without coronary artery disease.

Although the findings of this study are provocative, this study has important limitations. Recently, intensive regimens with 80 mg of atorvastatin or 20 mg of rosuvastatin have become available in Korea and produce greater reductions in atherosclerotic lipoprotein levels, which is particularly useful in patients with established coronary artery disease or acute coronary syndrome. Further studies comparing statins across dose ranges in patients not reaching their target goal with low-dose statins are required. Additionally, although changes metabolic parameters were not the primary endpoint of this study, a trend toward differences in blood glucose levels was observed between the two statins. Further studies are needed to elucidate the metabolic effects of statins.

In conclusion, this study demonstrated that rosuvastatin 10 mg is significantly more effective than atorvastatin 10 mg in reducing LDL-C levels in patients with nondiabetic metabolic syndrome, especially among those with lower NCEP ATP III target level goals. Both statins were well tolerated.

Conflict of interest

Acknowledgements

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