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ORIGINAL ARTICLE

Comparison of Rosuvastatin Versus Atorvastatin on Lipid Lowering Effectiveness in Hyperlipidemic Patients

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ABSTRACT

Hyperlipidaemia is the utmost important factors influencing coronary heart disease. Statins are supposed to be the 1st line of drug in clinical exercise for lowering low-density lipoprotein, total cholesterol and increasing HDL cholesterol. This study was held for the comparison of the safety and efficacy of atorvastatin and rosuvastatin in lowering hyperlipidaemia among hyperlipidemic patients.

Study Design: A prospective observational study.

Place and Duration: In the Medicine department of Mayo hospital, Lahore for three-months duration from 15th March 2021 to 15th June 2021.

Methods: This study comprised 90 patients with newly diagnosed hyperlipidaemia divided into two groups containing equal number of patients in both. Patients prescribed 10 mg of atorvastatin in Group A and 5 mg of rosuvastatin in Group B once daily for 6 weeks by the treating doctor of medicine. The data was saved in a personalized proforma format, and the SPSS 20.0 was used for analysis.

Results: Serum total cholesterol decreased significantly afterwards the treatment with the rosuvastatin and atorvastatin groups ($p < 0.00001$, correspondingly), but no significant variance ($p = 0.510$) noticed among the both groups treated with statins. The decrease in serum triglycerides was also significant ($p = 0.0006$ in the rosuvastatin group and $p = 0.042$ in the atorvastatin group) but the comparable difference in lowering hyperlipidemia was no significant among the both groups ($p = 0.309$). Similarly, LDL-C serum levels were significantly reduced in both groups ($p < 0.00001$) but no statistically significant variance ($p = 0.756$) noticed among the two groups in terms of efficacy.

Conclusion: No substantial changes in serum HDL levels were observed. The difference among the both groups was not significant ($p = 0.731$). This analysis = shows that both rosuvastatin and atorvastatin improved the lipid profile, but no changes were seen significantly among the both groups.

Keywords: Hyperlipidemia, Atorvastatin and Rosuvastatin.

INTRODUCTION

Hyperlipidaemia is an abnormal increase in serum lipids, result in the progression of atherosclerosis which is an arterial wall chronic disease and can lead to the sequelae of peripheral vascular disease, ischemic stroke and myocardial infarction[1-2]. Worldwide, 1/3rd of the coronary heart diseases is credited to high cholesterol, which is predictable to source 29.7 million disabilities and 2.6 million deaths[3-4]. Drugs that are hydroxymethylglutaryl coenzyme-A (HMG Co-A) reductase inhibitors are very effective in the treatment of dyslipidaemia and are therefore widely used in the treatment and inhibition of cardiovascular diseases. Statins inhibit cholesterol synthesis by reducing the synthesis of mevalonate, a direct product of HMG-CoA[5-6]. The statin group includes several drugs, including atorvastatin, and rosuvastatin is now widely used[7].

Many studies have shown an association between LDL-C cholesterol and high total cholesterol (TC) at the onset of CAD[8]. To avoid coronary heart disease, LDL-C and TC levels must be condensed < 116 mg / dL and 443.0 mg / dL, correspondingly. Statins are supposed to be the 1st line of drug in clinical exercise to lower low-density lipoproteins and total cholesterol to avert cardio-vascular disease[9-10]. This study was planned to compare the lipid-lowering efficacy of the promising new statin rosuvastatin with the currently widely used statin atorvastatin in patients with dyslipidaemia to guide current treatment strategies.

MATERIAL AND METHODS

This prospective observational study was conducted in the medicine department of Mayo hospital, Lahore for three-months duration from 15th March 2021 to 15th June 2021. This study included 90 patients; male and female, aged 20 to 75 years with newly diagnosed hyperlipidaemia divided into two groups containing equal number of patients in both. The selected subjects were randomised to receive atorvastatin 10 mg in Group A or rosuvastatin 5 mg in Group B daily for 6 weeks. The criteria for eligible patients selected for treatment with fasting triglycerides <400 mg / dL and fasting LDL-C> 160 mg / dL. The subjects received supplementary lipid-decreasing medicine, smoking, alcohol consumption, hypersensitivity to any of the statins, antioxidant vitamins (vitamin C, E, A), anti-inflammatory drugs, antiplatelet or anticoagulants drugs and pregnant and lactating women with impaired liver and kidney function, severe infection, or fatal disease were excluded. Baseline measurements included total cholesterol, serum triglycerides, LDL-C, HDL-C and after 8 weeks; follow-up investigation was done. Taking all measure of aseptic practice, blood(5ml) was drawn by puncture of a vein from the antecubital vein and stored in K3EDTA (anticoagulant) tube. With the help of centrifugation machine (3500 rpm for ten mints), separation of plasma was done and stored at -20 ° C until examination in a micropipette. It is recommended to take the drug before meals in the evening. The regularity of taking medications was confirmed by phone and compliance of the patients recorded on the sheet. The volunteers were counselled to report any side effects of medications administered throughout the study duration. Patients were instructed to follow a strictly restricted-fat diet. The data was saved and processed in a worksheet of the Microsoft Excel. Quantitative variables are articulated as mean ± SD. The two groups mean values variations were evaluated by unpaired and paired two-tailed Student's t-test. The significance level was adjusted so that the "p" value was less than 0.05.

RESULTS

Table-I demonstrate the main demographic features of all patients with hyperlipidaemia. There were 23 men and 22 women in Group A and 26 males and 19 females in Group B.

Based on demographic features, no significant variance among the rosuvastatin and atorvastatin treatment groups was noted. Before administration of atorvastatin and rosuvastatin,

serum cholesterol levels (mean ± SD) in this group were 260.2 ± 22.1 mg / dl and 265.1 ± 39.2 mg / dl. Later to 8 weeks of dosing, the same parameter changed to 160.1 ± 40.2 mg / dl in the atorvastatin group and 152.1 ± 34.1 mg / dl in the group of rosuvastatin (Table II).

Table I: Demographic characteristics of both groups before intervention

Characteristics	Atorvastatin Group (n= 45)	Rosuvastatin Group (n= 45)	p value
Age (years)	44.4 ± 11.2	41.8 ± 9.2	0.731
Sex			
Male	23	26	0.005
Female	22	19	0.005
Body weight (kg)	70.1 ± 10.2	64.2 ±9.2	1.121
Blood pressure			
Systolic (mmHg)	130.1 ± 15.9	133.3 ± 21.4	0.509
Diastolic (mmHg)	80.1 ± 10.1	86.4 ± 12.5	0.160
Hypertension	22	26	0.079
Diabetes mellitus	9	6	0.940

Table II: Effect on serum lipid profile

Variables (mg/dL)	Atorvastatin group (n = 45)				Rosuvastatin group (n = 45)				p value	P value
	Before	After	ap value	% Change	Before	After	p-value	% Change		
Total cholesterol	260.2 ± 22.1	160.1 ± 40.2	<0.00001	↓ 40.1	265.1 ± 39.2	152.1 ± 34.1	<0.00001	↓ 38.2	0.559	0.510
Triglyceride	200.1 ± 81.1	163.1 ± 72.1	0.042	↓ 18.2	196.5 ± 54.2	142.5 ± 63.0	0.0005	↓ 28.1	0.970	0.309
LDL-C	176.4 ± 19.2	90.8 ± 38.3	<0.00001	↓ 45.0	180.4 ± 31.9	87.1 ± 34.1	<0.00001	↓ 50.1	0.650	0.756
HDL-C	41.8 ± 10.2	37.5 ± 12.8	0.980	↓ 0.1	42.2 ± 8.1	41.5 ± 10.2	0.701	↓ 1.6	0.480	0.718

This alteration was significant statistically (P<0.0001, respectively), but after the treatment; the difference among the two groups was not significant (p = 0.510, Table II). The mean (%) reduction in serum cholesterol was 40.1% in the group of atorvastatin and 38.2% in the rosuvastatin group (Table II). At baseline, serum triglyceride concentrations (mean ± SD) were 200.1 ± 81.1 and 196.5 ± 54.2 mg / dL for the atorvastatin and rosuvastatin treatment groups. After 8 weeks of dosing, the serum triglyceride concentration changed to 163.1 ± 72.1 and 142.5 ± 63.0 mg / dL in the atorvastatin and rosuvastatin groups, correspondingly (Table II) and alteration was statistically significant (p = 0.042 and 0.0005, respectively) but later to the treatment, no significant variance was noted among the 2 groups (p = 0.309; Table II). Prior to administration of atorvastatin and rosuvastatin, serum LDL-C levels in each group were 176.4 ± 19.2 and 180.4 ± 31.9 mg / dl. After 8 weeks of dosing, the same parameter changed to 90.8 ± 38.3 mg / dL in the atorvastatin group and 87.1 ± 34.1 mg / dL in the rosuvastatin group, respectively (Table II) and no significant alteration among the groups after the treatment (p = 0.756; Table II).

At baseline, serum HDL-C concentrations (mean ± SD) were 41.8 ± 10.2 and 42.2 ± 8.1 mg / dL in the atorvastatin and rosuvastatin groups. After 8 weeks of dosing, serum HDL-C levels were 37.5 ± 12.8 and 41.5 ± 10.2 mg / dL in the atorvastatin and rosuvastatin groups, correspondingly (Table II). Again, no significant change was noticed among the both groups after the treatment (p = 0.701). The mean (%) reduction in serum HDL-C was 0.1% in the atorvastatin group and 1.8% in the rosuvastatin group. In this study, both rosuvastatin and atorvastatin were tolerated well by the patients. Three patients in the group of atorvastatin have complaint of headache and abdominal discomfort, and constipation was noticed in one patient in the rosuvastatin group. There were no grave side effects in the two groups that required dose adjustment or treatment discontinuation.

DISCUSSION

Data from the meta-analysis of VOYAGER suggest that every dosage of rosuvastatin is equal to a 3- to 3.5-fold dose of atorvastatin relative to the reduction in LDL-C[11-12]. This indicates that rosuvastatin 5 mg corresponds to atorvastatin 15-20mg. In this study, a beneficial consequence of rosuvastatin and atorvastatin on the serum lipid profile was observed. Based on previous study results, both decrease triglycerides, serum total cholesterol and LDL-C levels after drug treatment significantly[13-14]. A three-months analysis exhibited that both rosuvastatin (10 mg) and rosuvastatin (5 mg) significantly decreased LDL-C and total cholesterol compared with patients treated with 10 mg atorvastatin[15]. Another 8-week study of 10 mg rosuvastatin treatment significantly decrease LDL-C and triglycerides in comparison to 10 mg atorvastatin[16-17]. This suggests that compared to 10 mg of atorvastatin, 5 mg of rosuvastatin may be insufficient to significantly improve the lipid profile. There was no significant increase in HDLC levels in this study[18]. However, a slight decrease in HDL-C levels was observed in both groups. This is similar to the previous study with atorvastatin and rosuvastatin, which showed a slight decrease in serum HDL-C after 4 weeks of treatment. A similar study showed a decrease in HDL-C in the first 6 weeks, followed by an upsurge in HDL-C after sixteen weeks of intervention[19-20]. In this study, it is expected that longer than 8 weeks may be required to successfully demonstrate the outcome of statins on serum HDL-C levels[21]. In this study, both rosuvastatin and atorvastatin were highly operative in decreasing serum LDL-C in hyperlipidaemic patients. When the serum lipid lowering effect was measured as a proportion, rosuvastatin showed a greater improvement than that of atorvastatin but both have comparable safety profile[22-23]. So, rosuvastatin is a better treatment option than atorvastatin in lowering serum LDL-C in patients with hyperlipidaemia[24].

CONCLUSION

Both rosuvastatin and atorvastatin successfully improved the lipid profile in hyperlipidaemic patients. No significant variation was noticed among the both groups, although the effects of rosuvastatin seemed to be better given the percentage changes.

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