ORIGINAL RESEARCH

Assessment of efficacy of atorvastatin and rosuvastatin on various aspects of lipid profile

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ABSTRACT

Background: Dyslipidemia is inseparably associated with atherosclerosis - the widely prevalent pathological process, which is undoubtedly the cause of the most fatal and disabling disorders in man in current times. The present study was conducted to compare the efficacy of atorvastatin and rosuvastatin on various aspects of lipid profile.

Materials & Methods:100 patients with abnormal lipid profile attending the OPD/Wards of Department of Medicine, Guru Nanak Dev Hospital, attached to Government Medical College, Amritsar (Pb)were randomly divided into 2 groups of 50 each, Group A and Group B. Group A was put on Atorvastatin 20mg daily and Group B was put on Rosuvastatin 20mg daily.

Results: Out of 50 patients, males were 26 and females were 24. The mean ±S.D. values of various parameters of 100 patients in Group A and Group B. In group A serum total cholesterol, Serum Triglycerides, VLDL-C, LDL-C and HDL-C were 256.54±33.79 mg%, 284.52±72.26 mg%, 56.90±14.45 mg%, 160.25±31.09 mg% and 39.38±5.17 mg% respectively whereas in group B these values were 244.82±32.52 mg%, 250.18±38.20 mg%, 50.03±7.65 mg%, 158.08+31.63 mg% and 36.71 ±2.75 mg% respectively. The difference was significant (P< 0.05). There were 5 cases in group A and 4 in group B having dyspepsia, 1 case in group A and 2 cases in group B of myalgia.

Conclusion: Both Atorvastatin and Rosuvastatin were comparable in dyslipidemic groups.

Key words: Atorvastatin, Dyslipidemia, Rosuvastatin

Introduction

Dyslipidemia is inseparably associated with atherosclerosis - the widely prevalent pathological process, which is undoubtedly the cause of the most fatal and disabling disorders in man in current times; both have a close relation right from initiation to the final stages leading to clinical events.[1] It is a heterogenous group of disorders of lipid transport that result from accelerated or retarded degradation of lipoproteins that transport cholesterol and triglycerides through plasma and consists of excessive accumulation or lowered levels of one or more of major lipids transported in plasma and is a manifestation of one or more abnormalities of metabolism of transport.[2]

Dyslipidemia mainly manifests with two life threatening abnormalities: Accelerated Atherosclerosis and Acute Pancreatitis. Other manifestations include Xanthomas (Tendon, palmer, eruptive, tuboeruptive), Lipaemia Retinalis and Acute Abdominal Crisis.Atherosclerosis of coronary arteries may manifest as myocardial infarction and angina pectoris and that of central nervous system as non- haemorrhagic cerebrovascular accidents and transient ischaemic attacks.[3] In peripheral circulation it may cause intermittent claudication and gangrene and can render the limb disabled. Splanchnic involvement may cause mesenteric ischaemia. It may affect kidney as renal artery stenosis or as a frequent site of Atheroembolic disease.[4]Atherosclerosis remains the major cause of death and premature disability in industrialised countries and assuming importance as public health problem in developing countries like India. Coronary Artery Disease accounts for 12 million deaths annually over the globe[6]. Current predictions

estimate that by2020 A.D, cardiovascular disease will become the leading global cause of total disease burden, defined as the years subtracted from health life by disability or premature death.[5]The present study was conducted tocompare the efficacy of atorvastatin and rosuvastatin on various aspects of lipid profile.

Materials & Methods

This study was conducted on 100 patients with abnormal lipid profile attending the OPD/Wards of Department of Medicine, Guru Nanak Dev Hospital, attached to Government Medical College, Amritsar (Pb). All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. The patients were randomly divided into 2 groups of 50 each, Group A and Group B. Group A was put on Atorvastatin 20mg daily and Group B was put on Rosuvastatin 20mg daily. All the blood samples were collected after minimum of 12 hours of overnight fasting. At each visit, five (5) ml blood sample was collected from the antecubital vein of right arm of the patients with patient in sitting posture, using disposable syringe and needle (24G) without applying tourniquet. The blood from the syringe was then transferred into the sterile test tube after removing the needle. The blood samples were then centrifuged in a centrifuge machine for separating the serum. The serum was then used for performing various estimations. The optical densities of the various solutions were read using calorimeter. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

Results

Table I: Distribution of patients

Total- 50		
Gender	**Male**	**Female**
Number	26	24

Table I shows that out of 50 patients, males were 26 and females were 24.

Table II: Various parameters of 100 patients

Parameters / Individuals	**Serum Lipids (Mean+SD)**		**Serum Lipoproteins (Mean±SD)**		
	Serum Cholesterol (mg/dl)	Serum Triglycerides (mg/dl)	Serum VLDL-C (mg/dl)	Serum LDL-C (mg/dl)	Serum HDL-C (mg/dl)
Total patients (n=100)	250.68±33.52	267.35±60.03	53.47±12.01	159.16±31.22	38.05±4.34
Group A (n=50)	256.54±33.79	284.52±72.26	56.90±14.45	160.25±31.09	39.38±5.17
Group B (n=50)	244.82±32.52	250.18±38.20	50.03±7.65	158.08±31.63	36.71±2.75

Table II gives the mean ±S.D. values of various parameters of 100 patients in Group A and Group B. In group A serum total cholesterol, Serum Triglycerides, VLDL-C, LDL-C and HDL-C were 256.54±33.79 mg%, 284.52±72.26 mg%, 56.90±14.45 mg%, 160.25±31.09 mg% and 39.38±5.17 mg% respectively whereas in group B these values were 244.82±32.52 mg%, 250.18±38.20 mg%, 50.03±7.65 mg%, 158.08+31.63 mg% and 36.71 ±2.75 mg% respectively. The difference was significant (P< 0.05).

Table III Assessment of side effects

Individuals / **Side effects**	**Group A (n=50)**	**Group B (n=50)**	**P value**
Dyspepsia	5	4	0.92
Myalgia (Mild muscle aches and Tiredness)	1	2	0.05
Increase in alanine transaminase levels (ALT)	0	0	0

Table III shows that there were 5 cases in group A and 4 in group B having dyspepsia, 1 case in group A and 2 cases in group B of myalgia.

Discussion

Statins are the most commonly prescribed lipid lowering agents because they are effective, well tolerated and easy to administer with a low risk of adverse drug reactions (<0.1 %) and few drug interactions.[6] Statins exert their major effect-reduction of LDL levels-through a mevalonic acid-like moiety that competitively inhibits HMG-CoA reductase. They affect blood cholesterol levels by inhibiting hepatic cholesterol synthesis, increasing the synthesis of LDL receptors.[7] Degradation of LDL receptors also is reduced. The greater number of LDL receptors on the surface of hepatocytes results in increased removal of LDL from the blood, thereby lowering LDL-C levels. Statins also can reduce LDL levels by enhancing the removal of LDL precursors (VLDL and IDL) and by decreasing hepatic VLDL production.[8]The present study was conducted to compare the efficacy of atorvastatin and rosuvastatin on various aspects of lipid profile.

We found that out of 50 patients, males were 26 and females were 24. Schwartz et al[9]compared Rosuvastatin and Atorvastatin for reducing serum LDL-C in adults with hypercholesterolemia, at 24 weeks, serum LDL-C was reduced significantly more with Rosuvastatin 80 mg than Atorvastatin 80 mg (60% v/s 52%o, p<0.001). At 12 weeks Rosuvastatin 5 mg and 10 mg reduced serum LDL-C significantly more than Atorvastatin 10 mg (40%, p<0.01; 47%o, p<0.001 v/s 35%). At 18 weeks, serum LDL-C reductions were also significantly greater in both Rosuvastatin groups than in Atorvastatin group (52%, p<0.01; 59%, p<0.001 v/s 47%). Consequently, more patients receiving Rosuvastatin achieved serum LDL-C goals. Serum total cholesterol, serum HDL-C, serum non- HDL-C, apo lipoprotein-B and Aj and all lipid ratios were more favourably modified by Rosuvastatin at 24 weeks (p<0.01). Effects of these against serum triglycerides were similar.

We observed that the mean ±S.D. values of various parameters of 100 patients in Group A and Group B. In group A serum total cholesterol, Serum Triglycerides, VLDL-C, LDL-C and HDL-C were 256.54±33.79 mg%, 284.52±72.26 mg%, 56.90±14.45 mg%, 160.25±31.09 mg% and 39.38±5.17 mg% respectively whereas in group B these values were 244.82±32.52 mg%, 250.18±38.20 mg%, 50.03±7.65 mg%, 158.08+31.63 mg% and 36.71 ±2.75 mg% respectively.Schuster[10]atherosclerosis with or without diabetes mellitus were randomized to open label Rosuvastatin 10 mg, Atorvastatin l0mg, 20 mg, Simvastatin 20 mg or Pravastatin 40 mg for 8 weeks. These patients either received these for another 8 weeks or shifted from Atorvastatin 10 mg, Simvastatin 20 mg and Pravastatin 40 mg to Rosuvastatin 10 mg or from Atorvastatin 20 mg to Rosuvastatin l0 mg or 20 mg. At the end of 8 weeks Rosuvastatin 10 mg enabled statistically significant more patients to achieve the Joint European serum LDL-C goals than other statins. At 16 weeks, a statistically significant greater percentage of patients who were switched to Rosuvastatin 10 mg achieved the Joint European serum LDL- C goal, compared with patient remaining on Atorvastatin 10 mg, Simvastatin 20 mg or Pravastatin 40 mg. The rate of ATP-III serum LDL- C goal achievement statistically favoured Rosuvastatin over other treatments. At 8 weeks, Rosuvastatin 10 mg reduced serum LDL-C by 47% compared with reduction of 37.2%, 43.7%, 35.4% and 31% respectively in patients treated with Atorvastatin 10 mg, 20 mg, Simvastatin 20 mg, Pravastatin 40 mg (p <0.001 for all). Significant reduction in serum total cholesterol with Rosuvastatin 10 mg were 32.5% versus 25.8%>, 30.9%, *24.3%* and 20.7% respectively (p <0.001) for all except p <0.01 v/s Atorvastatin 20 mg. Serum HDL-C was increased by 9.2% with Rosuvastatin 10 mg, 6.8% with Atorvastatin 10 mg (p <0.01), 5.7% with Atorvastatin 20 mg (p <0.001), 8% with Simvastatin 20 mg (p = N.S) and *7.6%* with Pravastatin 40 mg (p = N.S). Serum triglycerides were reduced by 18.9% with Rosuvastatin 10 mg v/s 15.9%, *18.3%,* 13.5% and 10.5% respectively by Atorvastatin 10 mg and 20 mg (p = N.S), Simvastatin 20 mg (p <0.01), Pravastatin 40 mg (p <0.001).

We found that there were 5 cases in group A and 4 in group B having dyspepsia, 1 case in group A and 2 cases in group B of myalgia. Strandberget al[11]compared the efficacy of treatment with Rosuvastatin l0 mg, 20 mg or Atorvastatin l0 mg, 20 mg in African Americans over 6 weeks. The reduction in serum LDL-C with Rosuvastatin 10 mg and 20 mg (37% and 46% respectively) was significantly greater than Atorvastatin (32%) and 39%). Significantly greater reduction in serum total cholesterol and serum non- HDL-C and significantly greater increase in serum HDL-C were also seen with Rosuvastatin. A greater percentage of patients on both doses of Rosuvastatin achieved NCEP ATP-III serum LDL-C goals.

Conclusion

Authors found that both Atorvastatin and Rosuvastatin were comparable in dyslipidemic groups.

References

1. Pasternak, R.C., Smith, S.C., Jr., Bairey-Merz, C.N., et al ACC/AHA/NHLBI Clinical Advisory on the Use and Safety of Statins. Circulation, 2002, 705:1024-1028.
2. Paoletti R, Fahmy M, Mahla G, Mizan J, Southworth H. Rosuvastatin demonstrates greater reduction of low density lipoprotein cholesterol compared with Pravastatin and Simvastatin in hypercholesterolemia patients: A randomized double-blind study. J Cardiovasc Risk 2001; 8: 383-390.
3. Olsson AG, Istad H, Luurilla O, Ose L, Stender S, Tuomilehto J et al: Effects of Rosuvastatin and Atorvastatin compared over 52 weeks of treatment in

patients with hypercholesterolemia. Am Heart J 2002; 144: 1044-1051.
4. Davidson M, Ma P, Stein EA, Gotto AM Jr, Raza A, Chitra R et al. Comparison of effects on low-density lipoprotein cholesterol and high-density lipoporotein cholesterol with Rosuvastatin versus Atorvastatin in patients with type Ha or lib hypercholesterolemia. Am J Cardiol 2002; 89: 268-275.
5. Brown WV, Bays HE, Hassman DR, McKenney J, Chitra R, Hutchinson H et al. Efficacy and safety of Rosuvastatin compared with Pravastatin and Simvastatin in patients with hypercholesterolemia: A randomized, double-blind, 52-week trial. Am Heart J 2002; 144: 1036-1043.
6. Jones PH, Davidson MH, Stein EA, Bays HE, McKenney JM, Cain VA et al. Comparison of the efficacy and safety of Rosuvastatin versus Atorvastatin, Simvastatin, and Pravastatin across doses (STELLAR Trial). Am J Cardiol 2002; 93: 152-160.
7. Schneck DW, Knopp RH, Ballantyne CM, McPherson R, Chitra RR, Simonson SG. Comparative Effects of Rosuvastatin and Atorvastatin across their dose ranges in patients with hypercholesterolemia and without active arterial disease. Am J Cardiol 2003; 91: 33-41.
8. Shepherd J, Huminghake DB, Barter P, McKenney JM, Hutchinson HG. Guidelines for lowering lipids to reduce coronary heart disease risk: a comparison of Rosuvastatin with Atorvastatin, Pravastatin and Simvastatin for achieving lipid-lowering goals: American Journal of Cardiology 2003; 91(Suppl 5 A): 11C-17C.
9. Schwartz GG, Bolognese MA, Tremblay BP, Caplan R, Hutchinson H, Raza A et al. Efficacy and Safety of Rosuvastatin and Atorvastatin in patients with hypercholesterolemia and a high risk of coronary heart disease: A randomized controlled trial: American Heart Journal 2004; 148(1): e4.
10. Schuster H, Barter PJ, Stender S, Cheung RC, Bonnet J, Morrell JM et al. Effects of switching statins on achievement of lipid goals: Measuring effective reduction in cholesterol using Rosuvastatin therapy (MERCURY-I) study. American Heart Journal, 2004; 147(4): 705-712.
11. Strandberg TE, Feely J, Sigurdsson EL. Dirct statin comparison of Low-density Lipoprotein cholesterol values: An evaluation of rosuvastatin therapy compared with atorvastatin (DISCOVERY) study, ClmTher 2004; 26: 1821-1833.