COMPARING THE SAFETY AND EFFICACY OF ROSUVASTATIN AND ATORVASTATIN IN PATIENTS WITH CORONARY HEART DISEASES

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ABSTRACT

BACKGROUND

Heart disease is the leading cause of death in men and women in India and lowering LDL-C levels has become a cornerstone of heart health. When diet and exercise don't work, statins- drugs that interfere with the production of cholesterol can be simplest and cheapest treatment.

METHODS

The present comparative study was conducted in the Department of Cardiology. Total 60 cases were selected according to inclusion and exclusion criteria. And the cases were subdivided into two groups Group A and Group B. Group A consisting of 30 patients received Rosuvastatin (10) mg orally once daily and Group B consisting of 30 patients who will receive Atorvastatin (10) mg orally once daily for 12 weeks. Patient's blood sample will be collected to measure baseline lipid profile. Parameters of baseline were evaluated after 12 weeks of therapeutic intervention. Statistical analysis was done using the paired t test for comparing the lipid profiles of the two groups before and after treatment.

RESULTS

The results showed that Rosuvastatin to be better than Atorvastatin in terms of efficacy in improving the lipid profile, and both are equally safe.

CONCLUSIONS

Rosuvastatin proved to be better than Atorvastatin in terms of efficacy and Rosuvastatin and Atorvastatin are equally tolerated and equally safe.

KEYWORDS

Rosuvastatin, Atorvastatin, Lipid Profile

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BACKGROUND

Atherosclerosis is a major causal factor in the development of ischemic heart diseases. Ischemic cardiovascular and cerebrovascular events are the leading causes of morbidity and mortality.[1] The hypolipidaemic drugs have attracted considerable attention because of their potential to prevent cardiovascular disease by retarding the accelerated atherosclerosis in hyperlipidaemic patients.[2] Raised plasma CH is a major risk factor for coronary artery disease (CAD); higher the CH level, greater is the risk of CAD. Abundant data has confirmed that lowering the level of LDL-CH, results in lowering of cardiovascular mortality and morbidity. More recent evidence has indicated that prophylactic use of a statin in CAD/hypertensive patients even with average or lower than average CH levels lowers coronary and stroke events. With the availability of effective, well tolerated and safe hypolipidaemic drugs, it has become a standard practice to prescribe statin therapy after an acute coronary event irrespective of lipid levels.[3] Evidence that elevated plasma TG level or low plasma HDL-CH level poses independent high risk of CAD and stroke is also quite strong now.[4] Whereas raised LDL-CH is atherogenic, a higher HDL-CH level is either itself protective or indicates a low atherogenic state.[5]

The key measure in the lipid profile is LDL-C. Treatments that reduce LDL-C have been shown to reduce CHD risk by 25% to 45% over 5 years. An easy clinical tool to determine the elevated small dense LDL is the triglycerides/HDL ratio. According to a study in Indians, triglycerides/HDL ratio >3.0 could serve as a surrogate marker of small dense LDL in Asians.

The concentration of high-density lipoprotein cholesterol (HDL-C) is inversely related to CHD risk; HDL-C level of less than 40 mg/dl is considered a CHD risk factor. Low HDL-C is often a marker for other risk factors, including increased remnant lipoproteins; obesity; insulin resistance; diabetes; physical Inactivity; and genetic disorders.

Triglyceride levels >200 mg/dl increases the risk of CHD. Very high triglyceride levels (i.e., >500 mg/dl) indicate

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the presence of chylomicrons in addition to VLDL particles. Patients with very high triglyceride levels, especially those in whom triglycerides exceed 1,000 mg/dl, are at increased risk for pancreatitis. The relationship between cholesterol levels and CHD risk is continuous over a broad range.

METHODS

Inclusion Criteria

Men & women aged 21-75 years with a previous history of acute MI or diabetes mellitus are taken into study lipid levels in the range of: TC<240 mg/dl; TG<350 mg/dl, LDL-C 115-155 mg/dl, were selected for the study.

Exclusion Criteria

Subjects with history of serious hypersensitivity reactions to statins, history of any malignancy, current active liver disease, unexplained creatinine kinase levels, serum creatinine >2 mg/dl, uncontrolled hypothyroidism, history of alcohol or drug abuse in the last 5 years and Women who are pregnant or breast feeding or child bearing age are excluded from this study.

Informed consent is taken from the subjects participating in the study and clinical history was collected through a structured questionnaire. Fasting blood samples were collected from Control and study group subjects and suitable anticoagulant was added and plasma was separated and used for further analysis. Total cholesterol was estimated by CHOD-PAP method. Triglycerides were measured by GPO method. HDL-C was measured by Phosphotungstic acid method. Results were obtained from ERBA CHEM 7 Semi autoanalyzer. VLDL was calculated by Friedwald equation.[6] Results were expressed as mean ± SD, before and after treatment the parameters were again measured by paired student 't' test. T and p value are calculated and 0.05 are considered as statistically significant. SGOT & SGPT were measured by kinetic mode by semi auto analyser.

RESULTS

Subjects in group A received Rosuvastatin and subjects in group B received Atorvastatin.

Group	Total Cholesterol (mg/dl)	
	Baseline (0 wks.) (Mean ± SD)	After 12 wks. (Mean ± SD)
GROUP A	220.20 ± 9.4	193.39 ± 9
GROUP B	224.97 ± 9.2	203.21 ± 7.9
	t value = 1.980 p value =0.0524 Not Significant(NS)	t value=4.475 P value <0.001 Significant (S)

Table 1. Mean Total Cholesterol in the Two Groups

Group	Triglyceride Levels (mg/dl)	
	Baseline (0 wks.) (mean ± SD)	After 12 wks. (mean ± SD)
Group A	312.87±29.54	281.64±25.02
Group B	315.52±18.06	295.72±17.05
	t value = 0.419 p value<0.676 Not significant (NS)	t value=2.546 p value=0.0136 significant (S)

Table 2. Mean Triglyceride Levels in the Two Groups

Group	HDL-C Levels (mg/dl)	
	Baseline (0 wks.) (mean ± SD)	AFTER 12 wks. (mean ± SD)
Group A	28.87±7.492	33.8±7.874
Group B	27.33±6.075	29.436±6.669
	t value = 0.8745 P value = 0.3855 Not significant (NS)	t value=2.316 P value=0.0241 Significant (S)

Table 3. Mean HDL-C Levels in the Two Groups

Group	LDL-C Levels (mg/dl)	
	Baseline (0 wks.) (Mean ± SD)	After 12 wks. (Mean ± SD)
Group A	132.49±6.6	112.05±4.543
Group B	134.373±4.828	116.49±4.507
	t value = 1.261 P value = 0.2123 Not significant (NS)	t value=3.8 P value=0.0003 Significant (S)

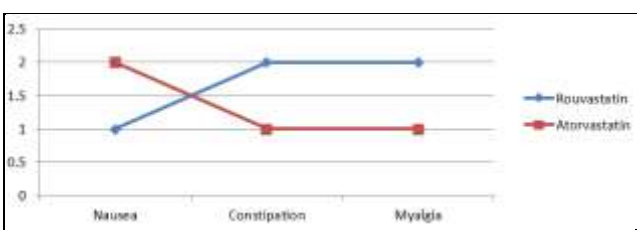
Table 4. Mean LDL-C Levels in the Two Groups

Group	SGOT (IU/l)	
	Baseline (0 wks.) (Mean ± SD)	After 12 wks. (Mean ± SD)
Group A	27.06±5.356	31.03±8.708
Group B	26.7±4.699	29.2±6.646
	t value = 0.2819 P value = 0.7790 Not significant (NS)	t value=0.9167 P value=0.3631 Not Significant (NS)

Table 5. Mean SGOT Levels in the Two Groups

Group	SGPT (IU/l)	
	Baseline (0 wks.) (Mean ± SD)	After 12 wks. (Mean ± SD)
Group A	22.1±6.216	26.3±7.910
Group B	20.96±5.36	25.13±7.33
	t value = 0.7638 P value = 0.4481 Not Significant (NS)	t value=0.5924 P value=0.5559 Not Significant (NS)

Table 6. Mean SGPT Levels in the Two Groups



Graph 1. Graphic Representation of Side Effects

DISCUSSION

Statins or HMG CoA reductase inhibitors are lipid lowering agents. They are now usually first drug of choice, especially because they are easy to use and have few serious side effects or contraindications.[7] According to many trails it was proved that Rosuvastatin controls cholesterol levels better when compared to other statins and it also improves the atherogenic and athero protective lipid profiles in patients with hypertriglyceridemia.[8] The results of the present study are compared with the previous studies regarding the efficacy of Rosuvastatin. In the present study, there is better reduction in TC, TG, LDL-C and increase in HDL-C levels by Rosuvastatin when compared with Atorvastatin and it is statistically significant (P<0.05).

The reduction in mean total cholesterol in patients on Rosuvastatin is 26.81 mg/dl i.e, from 220.20 mg/dl (0 wks.) to 193.39 mg/dl (12 wks.) and in patients in Atorvastatin is 21.76 mg/dl i.e, from 224.97 mg/dl (0 wks.) to 202.48 mg/dl (12 wks.) respectively. The percentage reduction in mean TC in group A and B are 12.17% and 9.6% respectively. This

suggests that patients on Rosuvastatin showed good reduction in TC levels when compared to Atorvastatin group which is statistically significant (P <0.001). Our results are comparable with previous studies. Michael Davidson et al has reported decrease in mean total cholesterol was more with Rosuvastatin compared with Atorvastatin (i.e., percentage decrease in TC was 30% and 25% respectively).[9] In another study by Michael B Clearfield et al, percentage decrease in mean TC attained at the end of the study Rosuvastatin group and Atorvastatin group is 30.8% (R= 10 mg) and 30.7% (A=20 mg) which has statistically significant difference.[10] Peter H Jones et al has reported decrease in mean TG was more with Rosuvastatin compared with Atorvastatin (i.e., percentage decrease in TG was 19.8% and 20% respectively).[11] Michael Davidson et al has reported decrease in mean LDL-C was more with Rosuvastatin compared with Atorvastatin (i.e., percentage decrease in LDL-C was 43% and 35% respectively).[12]

In another study by Faergeman O et al, percentage decrease in mean LDL-C attained at the end of the study Rosuvastatin group and Atorvastatin group is 47% (R= 10 mg) and 39% (A=10 mg) which has statistically significant difference.[12] In another study Peter H Jones et al, percentage decrease in mean LDL-C attained at the end of the study Rosuvastatin group and Atorvastatin group is 45.8% and 36.8% which has statistically significant difference.[11] In the present study the mean increase in the HDL-C levels in patients on Rosuvastatin is 4.93 mg/dl i.e., from 28.87 mg/dl (o weeks) to 33.8 mg/dl (12 weeks) and in patients on Atorvastatin is 2.10 mg/dl i.e, from 27.33 mg/dl (0 weeks) to 29.43 mg/dl (12 weeks) respectively.

In another study by Michael B Clearfield et al, percentage increase in mean HDL-C attained at the end of the study Rosuvastatin group and Atorvastatin group is 6.4% (R= 10 mg) and 3.1% (A=20 mg) which has statistically significant difference.[10] The most common adverse effects of statins are increase in serum transaminases, constipation, nausea and myalgia. At doses for reducing serum total cholesterol and LDL-C levels the side effects are mild and less. In the present study there were only 2 patients in group A (on Rosuvastatin) and 1 patient in group B(on Atorvastatin) with raised serum transaminases(SGOT and SGPT) which is clinically and statistically not significant. They were only 4 patients in group A (on Rosuvastatin) and only 5 patients in group B (on Atorvastatin) who complained of side effects like constipation, nausea and myalgia) which is clinically and statistically not significant. There were no differences in the incidence of side effects in both groups. Our results are comparable with previous studies like Michael Davidson et al and Shepherd et al found that Rosuvastatin and Atorvastatin has a similar safety profile when compared with other statins and demonstrated a favourable benefit risk profile across the dose range.[13]

CONCLUSIONS

Rosuvastatin proved to be better than Atorvastatin in terms of efficacy. Rosuvastatin and Atorvastatin are equally tolerated and equally safe.

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