DOI: 10.21276/aimdr.2019.5.6.ME6

Original Article ISSN (O):2395-2822; ISSN (P):2395-2814

Comparison of Atorvastatin and Fenofibrate in Dyslipidemia

Anuraag Jindal[1], Geetika Vohra[2], Satya B Nayyar[2]

[1]Consultant physician, Columbia Asia Hospital, Patiala.

[2]Senior Resident, Department of Pathology, Government Medical College, Patiala.

Received: October 2019
Accepted: October 2019

ABSTRACT

Background: Abnormalities in plasma lipoproteins and deranged lipid metabolism rank as most firmly established and best understood risk factor for atherosclerosis Lipid regulating drugs, may be prescribed if, dietary therapy and lifestyle modification fails to adequately normalize blood lipid levels. The European / Atherosclerosis Society and National Cholesterol Education Programme (NCEP) consider fibric acid derivatives (Fibrates) and Hydroxy Methylglutaryl CoA reductase inhibitors (HMG CoA reductase inhibitors) to be effective therapy for combined dyslipidernia . The aim of present study was to compare the efficacy of Atorvastatin and Fenofibrate on various aspects of lipid profile viz. total cholesterol, serum LDL-C, serum VLDL, serum HDL-C and serum triglycerides in Indian patients having dyslipidemia. **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. **Results:** The patients were randomly divided into 2 groups of 50 each, group A and B. Group A were put on Atorvastatin 10-20 mg daily and Group B were put on micronized Fenofibrate 200mg daily. **Conclusion:** The study concluded that none of these drugs were independently able to achieve NCEP ATP III goals. Atorvastatin has main effect or total serum cholesterol and LDL-C whereas Fenofibrate has main effect on serum triglycerides, VLDL-C and HDL-C .Combination of these may be tried to achieve the desired goal.

Keywords: Atorvastatin, Fenofibrate, Dyslipidemia.

INTRODUCTION

Atherosclerosis leading to coronary heart disease, ischaemic cerebrovascular disease and peripheral vascular disease accounts for the majority of morbidity and mortality among middle aged and older adults. Abnormalities in plasma lipoproteins and deranged lipid metabolism rank as most firmly established and best understood risk factor for atherosclerosis. The dyshpidemia comprise a heterogeneous group of disorders arising from alteration in normal plasma lipid and lipoprotein profiles.[1] Various classes of lipoprotein are total cholesterol, Very Low Density Lipoprotein Cholesterol (VLDL-C), Low Density Lipoprotein Cholesterol (LDLC), High Density Lipoprotein Cholesterol (HDL-C), High Denicity Lipoprotein Cholesterol (HDL-C), chylomicrons and triglycerides.[2] Risk reduction may be achieved by lowering triglycerides (TG) and raising high density lipoprotein cholesterol (HDL-C) levels 24. Lipid regulating drugs, may be prescribed if, dietary therapy and lifestyle modification fails to adequately normalize blood lipid levels. The European / Atherosclerosis Society and National Cholesterol Education Programme (NCEP) consider fibric acid derivatives (Fibrates) and HydroxyMethylglutaryl CoA reductase inhibitors (HMG CoA reductase inhibitors) to be effective therapy for combined dyslipidernia.[3]

Statins (HMG CoA reductase inhibitors) are most effective and tolerable agents for treating hypercholesterolemia. Various statins are simvastatin, pravastatin, lovastatin andatorvastatin 23. Various fibrates areclofibrate, gemfibrozil, bezafIbrate an fenofibrate.[1]

Name & Address of Corresponding Author
Dr Geetika Vohra,
Senior Resident,
Department of Pathology,
Government Medical College,
Patiala.

Aims and Objectives

The aim of present study is to compare the efficacy of Atorvastatin and Fenofibrate on various aspects of lipid profile viz. total cholesterol, serum LDL-C, serum VLDL, serum HDL-C and serum triglycerides in Indian patients having dyslipidemia

MATERIALS AND METHODS

It was a randomized open study. Following patients were excluded from the study

- Hypersensitivity to Atorvastatin/Fenofibrate

- Patients having diabetes mellitus
- Pregnancy/ Nursing mothers
- Active liver disease/Renal insufficiency! Nephroticsyndrome
- Uncontrolled hypothyroidism
- Alcoholic with> 21 drinks/week
- Acute MI within preceeding three months
- Known gastric acid disease/ peptic ulcer disease
- Pancreatitis/ cholelithiasis without cholecystectomy
- Concurrent intake of drugs affecting plasma lipidconcentration or known to interact with study medication (niacin, probucol, psyllium preparations, other fibrates, other HMG CoA reductase inhibitors, fish oils, immunosuppresants, steroids, isotretmion, cyclosporin, erythromycin)

This study began by recording lipid profiles in every patient at start and was repeated at four and eight weeks

Statistical Analysis

Data generated from the study was analysed according to standard statistical methods.

RESULTS

A total number of 100 patients of either sex, with combined dyslipidemia, were selected for present study. They were divided into 2 groups:
A: Atorvastatin - 50 patients,
B: Fenofibrate - 50 patients

Table 1: Baseline Characteristics of Subjects

Sex	Group A	Group B
Male	28	20
Female	22	30

Table 2: Mean Age (Years± S.D.)

Sex	Group A	Group B
Male	57.18±11.56	54.0±13.69
Female	55.54±9.18	53.38±10.81

Table 3: Coronary Artery Disease Risk Profile In Patients

	Hypertension	Smoking	Family History of Coronary arterydisease
Group A	33	8	14
Group B	37	3	17

Table 4: Values Of Various Parameters (Mean± Sd) In Mg% Of 100 Patients At Start Of Study

Mean± S.D	Total cholesterol (in mg%)	LDL-C (in mg%)	HDL-C (in mg%)	Triglyceride (in mg%)	VLDL-C (in mg%)
Total	252.66±	16097±	43.63±	277.73±	55.50±
Patients (n=100)	26.24	21.08	5.74	78.05	15.58
Group A (n=50)	25538± 25.85	16 V.3± 22.68	4506± 5.44	25606± 71.34	51-21± 14.27
Group B (n=50)	249.94± 26.60	161.56± 19.57	42.21± 5.74	299.40± 79.13	59.88± 15.83

Table 5: Serum Total Cholesterol (Mean± S.D) IN mg% AT 0,4 and 8 weeks

Group	0 Week	4 Weeks	8 Weeks
A (n=5O)	255.38±25.85	209.56±20.76	190.42±17.48
B (n =50)	249.94±26.60	223.32±22.89	209.34±20.70

Table 6: LDL-Cholesterol (Mean ±S.D) IN mg% AT 0,4 and 8 weeks

Group	0 Week	4 Weeks	8 Weeks
Group A (n=50)	160.38±22.68	113.97±14.19	98.36±12.13
Group B (n=50)	161.56±19.57	136.60±20.16	124.14±19.19

Group A patients were divided into 2 sub groups, Al and Group AII (31 patients were put on Atorvastatin 10 mg daily and 19 patients were put on Atorvastatin 20 mg daily. These drugs were given for 8 weeks in both groups. Lipid profile was done at 4 and 8 weeks.

Table 7: Serum Hdl-Cholesterol (Mean) in Mg% at 0,4, and 8 Weeks

Group	0 week	4 Weeks	8 Weeks
Group A (n=50)	45.06±5.44	46.52±5.71	48.31±5.74
Group B (n=50)	42.21±5.74	44.39±6.55	47.48±6.97

Table 8: Serum Triglycerides (Mean ±S.D) At 0,4, and 8weeks

Group	0 Week	4 Weeks	8 Weeks
Group A (n=50)	256.06±71.34	232.90±65.34	217.0±60.88
Group B (n=50)	299.40±79.13	211.58±64.81	188.88±56.28

Table 9: VLDL-Cholesterol (Mean ±S.D) in mg% at 0,4, and 8 weeks

Group	0 Week	4 Weeks	8 Weeks
Group A (n=50)	51.21±14.27	46.58±13.-07	42.76± 13.39
Group B (n=50)	59.88±15.83	42.32±12.96	37.78±11.26

Table 10: Side Effects

Side Effects	Group A	Group B
Dyspepsia	4	5
Myalgia (Mild muscle aches and tiredness)	1	1
Increase in Alanine transaminase levels	0	0
side Effects	**Group AI**	**Group AII**
Dyspepsia	1	3
Myalgia (Mild muscle aches and tiredness)	0	1
Increase in Alanine transaminase levels (ALT)	0	0

DISCUSSION

Coronary artery disease is a major problem in Indians and genetic factors appear to play an important role along with conventional and emerging risk factors. Since conventional risk factors do not explain the excess burden of Coronary artery disease, conventional approaches for prevention and treatment may be inadequate among Indians. It seems appropriate to begin aggressive preventive strategies directed at all risk factors at an earlier age in Indians. Abnormalities of the lipoproteins from both nature and nurture play a crucial role in the development of atherosclerosis. Use of different hypolipidemic drugs provides an effective way to reduce the risk of Coronary artery disease. The total cholesterol and LDL-C are undoubtedly the Coronary artery disease risk factors and their reduction has a beneficial effect on Coronary artery disease morbidity and mortality. LDLC is the primary target of lipid lowering therapy and sum of LDL and VLDL is the secondary target of therapy in patients with high triglycerides4 (ATP III Guidelines, 2002).

Two types of widely used lipid lowering drugs are-statins and fibrates. The present study was conducted to compare theefficacy of a statin (Atorvastatin) and a fibric acid derivative (Fenofibrate). 100 patients participated in the study. Apart from dyslipidemia other risk factors like hypertension, family history Coronary artery disease and smoking were also present. 31 patients were put on lipid lowering therapy for secondary prevention, rest 69% patients were put on drugs for primary prevention.

In this study the mean total cholesterol level at the start of the therapy was 252.66±26.24 mg%. This value is higher than that reported in other studies. Réddy et al,[17] reported mean total cholesterol of 196±37 mg/dl and Gopinath et al,[11] reported mean total cholesterol of 199 mg/dl. The mean LDL-C level the start of the therapy was 160.97±21.08 mg%. Gupta et al,[12] had reported mean LDL-C of 154.99±22.73mg%. The mean L-C level at the beginning of the study was 43.634±5.74 mg% A Reddy et al,[17] reported mean HDL-C of 43.9±7 mg%. The triglyceride levels at the beginning of the study were 77.3±78.05. Even this value is higher than that reported in otherstudies. Reddy et Al,[17] had reported mean triglycerides of 10.2± 45 mg%. Gupta et al,[12] reported mean triglyceride of 6.1±55 mg% in Jaipur. The differences in our study from previous studies may be due to difference in demographic profile of two populations. This suggests that changed dietary habit is responsible for rise in various lipoproteins over the years.

Effect of treatment with Atorvastatin

In the present study, treatment with Atorvastain for 8 week (Group A) resulted in statistically highly significant decrease in tote serum cholesterol and LDL-C by 25.15% and 38.14% respectively The reduction in total serum cholesterol and LDL-C between 0-weeks was 17.74% and 28.38% respectively and this value was statistically highly significant ($p<0.001$). These values show that the maximum reduction of total cholesterol and LDL-C occurred in firs four weeks and the reduction continued in next 4 weeks though i was comparatively less. These values were near to those reported by Ooi et al.[3] Who found a 27% and 30% decrease in total cholesterol and LDL-C respectively, in a 12 week study with Atorvastatin 10mg daily. Bairaktari et al,[6] reported respective decrease of 24.8% and 35.31% for similar dosages and time period. Schrott et al.[18] These observations suggested that the lipid reduction observed with Atorvastatin was rapid and may benefit the vascular endothelium.

The present study showed a statistically significant increase in HDL-C concentrations by 7.27% after 8 weeks treatment with Atorvastatin ($p<0.05$). This was less compared to 11% increase reported by Ooi et al,[3] after 12 weeks of therapy. Frost et al,[10] reported an increase of 10% after 6 weeks with similar dosages. Wang and Ting,[19] reported increase by 11% after 8 weeks therapy, whereas Winkler et al,[20] reported only 3% increase after 8 weeks therapy. This variation in HDL-C increase in various studies may be due to baseline HDL-C levels. More low the baseline HDL-C levels more is the increase after treatment (Branchi et al.[7] Serum triglyceride levels decreased significantly by 1:5.20% at the end of 8 weeks ($P<0.05$). The decrease was maximum in first 4 weeks of treatment. Increasing the dose of Atorvastatin from 10 to 20 mg in A did not cause any significant difference in triglyceride. This decrease in triglyceride levels was less as compared to25% reported by Ooi et al,[3] who gave Atorvastatin for 12 weeks. Bairaktari et al,[6] put similar dosage for weeks and resultant decrease in triglyceride levels was 12.5%. Wang and Ting,[19] reported triglycerides reduction by 23%. While Empken et al,[9] reported reduction in triglycerides of 9% on Winkler et al,[20] reported triglycerides reduction by 32%. The difference in triglyceride reduction among various studies can 1 due to different genetic makeup of various races in which the studies were done.

LDL-C levels decreased by 13.51% in 8 weeks and ti decrease was significant ($p<0.05$). This decrease was less than ti 35% reduction reported by Ooi et al,[3] after 12 weeks therapy. On increasing the dose of Atorvastatin from 10 to 20 mg rsignificant change was observed in VLDL-C levels.

Effect of treatment with Fenofibrate

In the present study, micronized Fenofibrate in dosage 200 mg daily was used for 8 weeks (Group B). Total cholesterol an L-C decreased by. 16.11% and 23.09% respectively ($p<0.05$).These values were

comparable to various previous studies. Bairaktari et al 6 reported decrease of 15.6% and 17.6% respectively after 12 week of treatment. Kiortsis et al,[16] reported 14.7% and 8.7% decrease in total cholesterol and LDL-C respectively after 6 weeks treatment.

Serum HDL-C levels increased significantly by 12.49%after 8 weeks of treatment with Fenofibrate (p<0.001). Ooi et al,[3] reported a 24% increase after 12 weeks of treatment. Bairaktariet al,[7] reported a 16.6% increase after 16 weeks of treatment. Zhu and Lee,[21] reported 12.7% increase in HDL-C vels after 8 weeks. The wide variations in HDL-C values may be due to baseline variations of HDL-C as the increase is more when baseline values are lower Branchi et al.[7]

Serum triglycerides and VLDL-C concentrations creased-significantly by 36.96% and 36.37% respectively <0.001). Bairaktari et al,[6] reported triglyceride reduction of % and Ooi et al,[3] reported reduction of VLDL-C by 56% after 12 weeks of treatment. Ceska and Stule,[8] reported 41. 1% reduction in triglyceride levels, Kiortsis et al,[16] reported 39.5% reduction in triglycerides after 6 weeks. Ducobu et al,[9] reported 38.7% reduction in triglycerides after 12 weeks treatment. The values in the present study are comparable to the observations in previous studies.

In the present study Atorvastatin caused a highsignificant reduction in total serum cholesterol (25.15%, p<0.001 and LDL-C (38.14%, p<0.001) on increasing dose of Atorvastatin from 10 mg to 20 mg, reduction in total cholesterol increased from 22.36% to 33.35% and reductions in LDL-C increased from 34.480,1 to 44.38% (both statistically significant, p<0.05). But this study had once again confirmed that even low doses of Atorvastatin are effective in decreasing in total cholesterol and LDL-C.

Fenofibrate (200 mg/day) had a significant but lesser impact on serum total cholesterol as compared to Atorvastatin (p<0.05). Effect on LDL-C concentration was significant (p<0.05) as it reduced the levels by 23.09%, though it was less as compared that of Atorvastatin.

Atorvastatin caused a lesser but still significant decrease in triglyceride concentration (15.20%, p<0.05). Reduction of VLDLby 13.51% was statistically significant (p<0.05). Maximal decrease in triglycerides was with Fenofibrate, which caused reduction36.96% (p<0.001) which was much more than impact Atorvastatin Similar effect occurred on VLDL-C, which decrease by 36.37% with Fenofibrate (p<0.001) in comparison to Atorvastatin which decreased VLDL-C by 13.51%, (p<0.05).

In patients with mild hypertriglyceridemia (triglyceride<400 mg%) LDL-C levels, usually decrease by 15%-20% with fibrates. In patients with more intense hypertriglyceridemia (>40 mg%) LDL levels can rise by 10-30% when put on fibrates.[22]

The present study has shown that both Atorvastatin and Fenofibrate has effect on all the lipoprotein fractions but the main effect of Atorvastatin is on serum total cholesterol and LDL-C whereas Fenofibrate has main effect on triglycerides, VLDL-C and HDL-C.

In the present study adverse drug reactions linked to drug therapy included the following:-

In Group A 4(8%) patients complained of Dyspepsia. 1(2%) patient complained of myalgia and no patient had rise in ALT levels.

In Group B 5 (10%) patients showed dyspepsia, 1(2%) patients showed myalgias and tiredness. No patient showed raised ALT level.

In the present study Atorvastatin in general was well tolerated. No patient had any serious side effect and no patientwithdrew from the study. GI tract disturbances were the main side effects occurring in 8% of patients. This side effect was dose related, with 10 mg/day of Atorvastatin only 3% patients had GItract disturbances and on increasing the dose to 20 mg/day itoccurred in 15.79% patients. This difference was statistically significant (p<0.05). This was in accordance with the study of Adkins and Faulds 1 in which GI tract disturbance was mostcommon side effect, occurring in 8.5% patients. This study also showed that side effects were dose dependent and increased with increases plasma drug levels. Similar observations were shared by Mahh and Bersot,[22] and Wang & Ting.[19]

CONCLUSION

At the beginning: of the study, all the patients had raised serum cholesterol (mean value 252.66±26.24 mg%) and/or raised LDL-C (mean value 160.97±21.08 mg%) low HDL-C (mean value 43.63± 5.74 mg%), raised triglycerides (mean value 277.73± 78.05 mg%) and raised VLDL-C (mean value-55.50± 15.58 mg%)

31 patients (31%) had evidence of Coronary artery disease and for them, the treatment was secondary prevention, 69 patients. (69%) had no evidence of Coronary artery disease and for them treatment was primary prevention Hypertension was observed in 70(70%) patients. Diabetes Mellitus was an exclusion criteria. In Group A, after giving Atorvastatin following observations we made.

1. Total serum cholesterol and LDL-C decreased by 25.15%(pcz0.001)and 38.14%(p<O.00respectively. Both were highly significant.
2. Rise in serum HDL-C was 7.27% (p<0.05).
3. Fall in serum triglycerides and VLDL-C was 15.20 and 13.51% respectively (p<z0.05, significant).
4. In Group A, out of 50 patients, 19 patients (380/c were given 20 mg of Atorvastatin. With increase i dose of Atorvastatin, there was significantly more decrease in total serum cholesterol and LDL(p<0.05) but, the effect on other parameters 1il HDL-C, triglycerides and VLDL-C was not (significant (p>0.05).

5. The maximum effect of Atorvastatin was observed infirst four weeks, the effect in subsequent four weekswas less as compared to the effect in first 4 weeks.

   In Group B, all 50 patients were put on micronisedFenofibrate 200 mg/day. The following observations were made:-

i. Serum triglycerides and serum VLDL-Cdecreased by 36.96% (p<O.00l) and 36.37% (p<0.001) respectively. Both reductions were highly significant.

ii. Serum HDL-C increased by 12.49% (p<0.001, highly significant).

iii. Serum total cholesterol and LDL-C decreased by 16.11% ard 23.09% respectively (p<0.05, significant).

With Atorvastatin (Group A) 8% patients complained ofGI tract disturbances, 2% had myalgias and none had rise inserum ALT levels. With Fenofibrate (Group B) GI tact side effects were observed in 10% and myalgias in 2%. No case had rise in ALT levels.

To conclude, none of these drugs were independently able to achieve NCEP ATP III goals. Atorvastatin has main effect or total serum cholesterol and LDL-C whereas Fenofibrate has main effect on serum triglycerides, VLDL-C and HDL-C Combination of these may be tried to achieve the desired goal. Further studies in this regard are needed.

REFERENCES

1. Adkins JC, Faulds D: Micronised Fenofibrate: A review of its Pharmacodynarnic Properties and Clinical Efficacy in the Management of Dyslipidaemia. Drugs 1997; 54(4): 61533.
2. MahleyRW, Bersot TP. ' Drug Therapy for Hypercholesterolemia and Dyslipidemia. In: Harman JG, Limbird LE, Gilman AG (editors). Goodman and Gilmans'. The Pharmacological basis of therapeutics 2001; The McGraw Hill Companies Inc, USA: 10: 971-1003.
3. OoiTC, Heinonen T, Alaupovic P, Davignon J, Leiter L, Lupien PJ et al. Efficacy and safety of a New Hydroxymethyiglutaryl Coenzyme A Reductase Inhibitor, Atorvastatin, in Patients with Combined Hyperlipidemia: Comparison with Fenofibrate. ArterioscierThrombVascBiol1997.; 17: 1793-1799.
4. ATP- III guidelines. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA, 2002; 285(19): 2486-2497.
5. ATP-II guidelines. Expert Panel on Detection, Evaluation and treatment of High blood cholesterol in Adults: Summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and Treatment of High Blood cholesterol in Adults (Adult Treatment Panel II). JAMA 1993; 269: 3015.
6. Bairakatrari TE, Tzalias CS, Tsimihodinios VK, Liberopoulos NE, Miltiadous GA, Elisaf MS. Comparison of the efficacy of Atorvastatin and micronisedFenofibrate in the treatment of mixed hyperlipidemia. Journal of Cardiovascular Risk 1999; 6: 113-116.
7. Branchi A, Fiorenza AM, Torn A, Muzio F, Berra C,.Colombo E et al. Atorvastatin increases HDL cholesterol in hypercholesterOlëmic patients. Evidence of a relationship with baseline HDL cholesterol.NutrMetabCardiovasc Dis 2002; 12(1): 24-8.
8. Ceska R, Stule T. Fenofibrate 250 SR Pharmavitin patients with mixed hyperlipoproteinemia. Results of a study of 189 patients from 13 centers in the Czech Republic.CasLekCesk 2000; 139(12): 369-73.
9. Ducobu J, VanHaelst L, Salomon H. Comparison of ronisedFenofibrate and pravastatin in patients with primary hyperlipidemia. Arch mt Med 2001; 149: 394.
10. Frost RJA, Otto C, Geiss HC, Schwandt P, Parhofer KG. Effects of Atorvastatin versus, Fenofibrate on lipoprotein profiles, low density lipoprotein subfraction distribution and hemorheologic parameters in type 2 Diabetes Mellitus with mixed hyperlipoproteinemia. Am J Cardiol 2001; 87: 44-48.
11. Gopinath N, Chadha SL, Sehgal A et al. What is desirablelipid profile.Indian Heart Journal 1994; 46: 325-27.
12. Gupta R, Deedwania PC, Soangra MR. Prevention of oronary heart disease in India: An epidemiological perspective. Indian Journal of Community Medicine 2002; XXVII: 4.
13. Gupta R, Gupta VP, Sarna M, Bhatnagar S, Thanvi J, Sharma V, et al. Prevalence of coronary heart disease and risk factors in an urban Indian population : Jaipur Heart Watch-2. Indian Heart J 2002; 54: 59-66.
14. Gupta R, Parkash H, Raul V. Cholesterol, lipoproteins, triglycerides, rural-urban difference and prevalence of dyslipidaemias among males in Rajasthan. J AssocPhysicans India 1997; 45: 275-279.
15. Gupta R. Prevention of Coronary Heart Disease in India.South Asian Journal of Preventive Cardiology 1998; 2: 41:51.
16. KiortsisN, Millionis H, Bairakatari El', Elisaf MS.of combination of Atorvastatin and micronized Fenofibrate in the treatment of severe mixedhyperlipidemia. J ClinPharmacol 2000; 56: 631-635.
17. Reddy KS, Pandit K, Nagtilak S Coronary risk factor profile of urban and rural population samples. Indian Heart Journal 1992; 44: 336.
18. SchrottH, Fereshetian AG, Knoop RH, Bays H, Jones PH, Littlejohn TW. A multicentrer, Placebo-controlled, Dose-Ranging study of .Atorvastatin. J CardiovascPharmacoiTher 1998; 3(2): 119-124.
19. Wang KY, Ting CT. A randomized, double-blind, placebo controlled, 8 week study to evaluate the efficacy and safety of once daily atorvastatin (10 mg) in patients with elevated LDL-cholesterol. Jpn Heart J 2001; 42(6): 725-38.
20. Winkler K, Welt P, Friedrich I, Schmitz H, NickellHH, Hauck P et al. Qualitative effect of Fenofibrate and quantitative effect of Atorvastatin on LDL profile in combined hyperlipidemia with dense LDL. ExpClinEndocrinol Diabetes 2004; 112(5): 241-7.
21. Zhuy J, Ye P. A survey on the efficacy and tolerability ofmicronisedFenofibrate in patients with dyslipidemia. Nature 2000; 301:709
22. Grndy SM: United States Cholesterol Guidelines •2001: Expanded Scope of Intensive Low-Density Lipoprotein-Lowering Therapy. Am J Cardiol 2001; 88: 23J-27J
23. Chong PH, Seeger JD, Franklin C: Clinically Relevant. . Differences between the Statins Implications for Therapeutic Selection.Am J Med 2001; 111: 390-400.
24. Mahley RW, Bersot TP: Drug Therapy for Hypercholesterolemia and Dyslipidemia. In: Harman JG, Limbird LE, Gilman AG (editors). Goodman and Gilmans'. The Pharmacological basis of therapeutics• 2001; The McGraw Hill Companies Inc, USA: 10: 9711003.

How to cite this article: Jindal A, Vohra G, Nayyar SB. Comparison of Atorvastatin and Fenofibrate in Dyslipidemia. Ann. Int. Med. Den. Res. 2019; 5(6):ME26-ME30.

Source of Support: Nil, **Conflict of Interest:** None declared