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Original Research Article

A Comparative Study on the Effectiveness of Atorvastatin and Atorvastatin plus Omega 3 Fatty Acid in patients with Dyslipidemia in a Tertiary Care Hospital

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Abstract

Introduction: Dyslipidemia is a major modifiable risk factor for cardiovascular disease and is characterized by abnormalities in lipid metabolism. Omega-3 fatty acids have demonstrated triglyceride-lowering properties and potential cardioprotective effects and may provide additional benefit when used in combination with statins. Therefore, the study aimed to compare the effectiveness of atorvastatin alone and atorvastatin plus omega-3 fatty acid in improving lipid profile parameters among patients with dyslipidemia attending a tertiary care hospital.

Materials and Methods: This prospective, randomized, open-label comparative study was conducted in a tertiary care hospital and included 60 newly diagnosed dyslipidemic patients aged 18–60 years. Participants were randomly allocated into two groups: Group A received atorvastatin 10 mg daily, and Group B received atorvastatin 10 mg daily plus omega-3 fatty acids 2 g/day for 12 weeks. Lipid profile parameters were measured at baseline and at 4, 8, and 12 weeks.

Results: Baseline demographic and clinical characteristics were comparable between the two groups. Both treatment regimens resulted in significant reductions in total cholesterol, triglycerides, LDL-C, and VLDL-C and a significant rise in HDL-C over 12 weeks ($p < 0.001$ within groups). There was no statistically significant difference between the two groups in total cholesterol, LDL-C, or HDL-C at any time point. However, triglyceride and VLDL-C reductions were significantly greater in the combination therapy group compared with atorvastatin alone.

Conclusion: Atorvastatin combined with omega-3 fatty acids provides superior triglyceride and VLDL-C reduction compared with atorvastatin monotherapy, with similar effects on total cholesterol, LDL-C, and HDL-C and a favorable safety profile.

Keywords: Dyslipidemia; Atorvastatin; Omega-3 fatty acids; Lipid profile; Triglycerides.

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Introduction

Dyslipidemia is a major modifiable risk factor for cardiovascular diseases and is characterized by abnormalities in lipid metabolism, including elevated total cholesterol, low-density lipoprotein cholesterol (LDL-C), triglycerides, and/or reduced high-density lipoprotein cholesterol (HDL-C) [1]. The global burden of dyslipidemia has increased steadily with changing lifestyles, dietary habits, urbanization, and reduced physical activity [2]. In India, the prevalence of dyslipidemia is rising rapidly, contributing significantly to the growing incidence of coronary artery disease and stroke. Early identification and effective management of lipid abnormalities are therefore essential for reducing long-term cardiovascular morbidity and mortality [3]. Statins remain the cornerstone of pharmacological therapy for dyslipidemia due to their proven efficacy in lowering LDL-C and reducing cardiovascular events [4]. Atorvastatin, a widely prescribed statin, acts by inhibiting HMG-CoA reductase and effectively reduces total cholesterol and LDL-C while modestly improving

HDL-C levels [5]. However, despite optimal statin therapy, many patients continue to exhibit residual lipid abnormalities, particularly elevated triglycerides and low HDL-C, which are independently associated with increased cardiovascular risk. This has prompted interest in combination therapy targeting multiple lipid fractions to achieve better lipid control [6].

Omega-3 fatty acids, primarily eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have been shown to reduce triglyceride levels through inhibition of hepatic very-low-density lipoprotein synthesis and enhanced clearance of triglyceride-rich lipoproteins [7]. In addition to their lipid-lowering effects, omega-3 fatty acids possess anti-inflammatory, antithrombotic, and endothelial-protective properties, which may contribute to cardiovascular risk reduction [8]. Combining omega-3 fatty acids with statins has been proposed as a rational therapeutic strategy, especially in patients with mixed dyslipidemia, to provide additive or synergistic effects on lipid parameters without significantly increasing adverse effects [9].

Although several studies have evaluated the benefits of statin monotherapy and combination therapy with omega-3 fatty acids, data comparing their effectiveness in routine clinical practice settings, particularly in the Indian population, remain limited [10]. Differences in dietary patterns, genetic predisposition, and comorbid conditions necessitate locally generated evidence to guide treatment decisions. Therefore, the study aimed to compare the effectiveness of atorvastatin alone and atorvastatin plus omega-3 fatty acid in improving lipid profile parameters among patients with dyslipidemia attending a tertiary care hospital.

Materials and Methods

This prospective, randomized, open-label comparative study was conducted in the outpatient department of the Department of General Medicine at Trichy SRM Medical College Hospital and Research Centre, Irungalur, Tiruchirappalli.

A total of 60 patients newly diagnosed with dyslipidemia were enrolled after obtaining written informed consent. Eligible participants were adults aged 18–60 years of either sex who met biochemical criteria for dyslipidemia, including elevated total cholesterol, LDL cholesterol, triglycerides, or low HDL cholesterol. Patients with pregnancy or lactation, chronic debilitating illnesses, known hypersensitivity to statins or fish products, deranged liver enzymes, jaundice, bleeding disorders, or significant renal or cardiac disease were excluded from the study.

Participants were randomly allocated into two groups of 30 each. Group A received atorvastatin 10 mg once daily, while Group B received atorvastatin 10 mg once daily along with omega-3 fatty acids at a dose of 2 g/day for a duration of 12 weeks. Each omega-3 capsule contained eicosapentaenoic acid (EPA) 270 mg and docosahexaenoic acid (DHA) 180 mg. Treatment was continued without dose modification throughout the study period. Medications were provided free of cost, and patients were instructed not to use any additional lipid-lowering drugs during the study. Both groups received standardized advice regarding dietary modification and physical activity.

Baseline evaluation included detailed clinical assessment and laboratory screening with hemoglobin, blood sugar, blood urea, serum creatinine, and electrocardiography. Lipid profile parameters (total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol, and VLDL cholesterol) were measured at baseline and subsequently at 4, 8, and 12 weeks. Liver function tests were performed at baseline and at the end of 12 weeks to assess hepatic safety.

The primary outcome measure was the change in lipid profile parameters from baseline to 12 weeks within each group and the comparison of mean changes between the two groups. Statistical analysis was performed using appropriate parametric tests to compare continuous variables between groups and within groups over time, and categorical variables were analyzed using the chi-square test. A p value of <0.05 was considered statistically significant.

Results

The two groups were comparable at baseline with respect to age distribution, mean age, gender, body weight, body mass index, and comorbidities. No statistically significant differences were observed between Group A and Group B for any baseline demographic or clinical variable, confirming homogeneity of the study population prior to intervention (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics (n = 60)

Parameter		Group A (n=30)	Group B (n=30)	p-value
Age (years)	20–30	2 (6.7)	2 (6.7)	0.986
	31–40	13 (43.3)	13 (43.3)	
	41–50	5 (16.7)	6 (20.0)	
	51–60	10 (33.3)	9 (30.0)	
	Mean ± SD	43.3 ± 9.63	43.4 ± 9.66	
Gender	Female	17 (56.7)	12 (40.0)	0.196
	Male	13 (43.3)	18 (60.0)	
Body weight (kg)	Mean ± SD	55.80 ± 9.90	56.00 ± 10.22	0.351
BMI (kg/m²)	Mean ± SD	26.57 ± 4.20	27.39 ± 3.45	0.681
Comorbidities	Diabetes	9 (30.0)	10 (33.3)	0.986
	Hypertension	5 (16.7)	5 (16.7)	
	Both	10 (33.3)	8 (26.7)	
	None	6 (20.0)	7 (23.3)	

Total cholesterol levels showed a significant decline over time in both groups ($p < 0.001$ for each group). However, there was no statistically significant difference between Group A and Group B at any time point, including baseline, 4 weeks, 8 weeks, and 12 weeks (all $p > 0.05$), indicating comparable cholesterol-lowering effects (Table 2).

Table 2: Total Cholesterol (mg/dL)

Time point	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value
Baseline	221.9 ± 9.7	226.9 ± 14.3	0.121
4 weeks	220.7 ± 8.6	224.5 ± 12.8	0.184
8 weeks	215.9 ± 7.5	215.1 ± 10.8	0.749
12 weeks	207.3 ± 10.7	207.8 ± 12.0	0.871
p-value	**p < 0.001**	**p < 0.001**	

Triglyceride levels decreased significantly over the study period in both groups ($p < 0.001$). Although Group B demonstrated a numerically greater reduction, the between-group differences at all time points were not statistically significant ($p > 0.05$) (Table 3).

Table 3: Triglycerides (mg/dL)

Time point	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value
Baseline	193.3 ± 26.8	212.8 ± 54.1	0.083
4 weeks	184.3 ± 19.7	195.6 ± 43.3	0.202
8 weeks	172.8 ± 17.3	174.9 ± 29.7	0.735
12 weeks	161.1 ± 16.0	154.6 ± 21.9	0.192
p-value	**p < 0.001**	**p < 0.001**	

HDL cholesterol levels increased progressively in both groups with statistically significant within-group changes over time ($p < 0.001$). The differences between Group A and Group B at all time points were not statistically significant, suggesting similar effects of both regimens on HDL levels (Table 4).

Table 4: HDL Cholesterol (mg/dL)

Time point	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value
Baseline	36.0 ± 4.9	36.9 ± 4.3	0.486
4 weeks	36.7 ± 4.7	38.0 ± 4.0	0.243
8 weeks	37.7 ± 4.6	38.9 ± 4.0	0.263
12 weeks	38.2 ± 4.5	39.9 ± 3.9	0.128
p-value	**p < 0.001**	**p < 0.001**	

LDL cholesterol levels declined significantly over time in both treatment groups ($p < 0.001$). There was no statistically significant difference between Group A and Group B at any time point, indicating equivalent LDL-lowering efficacy (Table 5).

Table 5: LDL Cholesterol (mg/dL)

Time point	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value
Baseline	141.2 ± 7.1	141.4 ± 8.6	0.909
4 weeks	141.2 ± 7.1	141.4 ± 8.6	0.922
8 weeks	137.6 ± 6.6	135.2 ± 9.2	0.239
12 weeks	132.4 ± 8.7	131.2 ± 11.1	0.652
p-value	**p < 0.001**	**p < 0.001**	

VLDL cholesterol levels showed a significant reduction across the study duration in both groups ($p < 0.001$). Intergroup comparisons at each time point did not reveal any statistically significant differences ($p > 0.05$) (Table 6).

Table 6: VLDL Cholesterol (mg/dL)

Time point	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value
Baseline	38.7 ± 5.4	42.6 ± 10.8	0.081
4 weeks	36.9 ± 3.9	39.1 ± 8.7	0.200
8 weeks	34.6 ± 3.5	35.0 ± 5.9	0.735
12 weeks	30.7 ± 4.2	30.7 ± 4.6	0.986
p-value	**p < 0.001**	**p < 0.001**	

At 12 weeks, AST and ALT levels were comparable between the two groups, with no statistically significant differences observed for either enzyme, indicating no differential hepatic effect between the treatment regimens (Table 7).

Discussion

Both groups were well matched at baseline for demographic and clinical variables, which strengthens attribution of observed lipid changes to the interventions rather than confounding characteristics. The pattern of comorbid diabetes and hypertension in a substantial subset of our cohort is consistent with prior reports of frequent clustering of cardiometabolic risk factors in patients with dyslipidemia, highlighting the clinical importance of addressing mixed lipid abnormalities in routine practice [11].

Our primary lipid findings, significant within-group reductions in total cholesterol, LDL-C, VLDL-C and triglycerides and an increase in HDL-C in both arms — are concordant with the literature showing robust lipid improvements with statin therapy and additional triglyceride lowering when omega-3 fatty acids are added. Meta-analyses and randomized trials have repeatedly shown that prescription omega-3 formulations (EPA ± DHA) produce clinically meaningful TG reductions, and when combined with statins they produce additive triglyceride and non–HDL reductions versus statin alone [11,12].

The significantly greater triglyceride and VLDL reductions observed in the atorvastatin + omega-3 group in our study mirror results from dedicated add-on trials (for example, prescription omega-3 agents added to background statin therapy) and pooled analyses that report larger TG declines with combination therapy compared with statin monotherapy. These data align with mechanistic expectations omega-3s reduce hepatic VLDL synthesis and enhance TG clearance and reinforce the role of omega-3s for patients with residual hypertriglyceridemia despite statin therapy. Notably, large outcome trials using high-dose EPA have also suggested clinical event benefit in selected high-risk, statin-treated populations, although outcome effects depend on formulation, dose, and patient selection [13,14]. Safety signals in our cohort were reassuring: no significant hepatic or hematological abnormalities and only transient, mild tolerability complaints in the omega-3 group. This safety profile is consistent with prior randomized studies and reviews showing that adding omega-3 formulations to statins does not materially increase rates of hepatic or muscular adverse events, although clinicians should remain mindful of dose-related risks and interpret outcome data in the context of specific omega-3 preparations [13,15]. Overall, our efficacy and safety observations are supported by contemporary evidence favoring omega-3 supplementation as a well-tolerated adjunct to statins for targeted lipid optimization, particularly for triglyceride-rich dyslipidemia.

Conclusion

In the present study, both atorvastatin monotherapy and combination therapy with atorvastatin plus omega-3 fatty acids produced significant improvements in lipid profile over 12 weeks. The addition of omega-3 fatty acids resulted in a significantly greater reduction in triglycerides and VLDL cholesterol compared with atorvastatin alone, while reductions in total cholesterol and LDL cholesterol and the rise in HDL cholesterol were comparable between the two regimens. Both treatments were well tolerated without significant adverse effects on liver enzymes or hematological parameters. These findings indicate that omega-3

fatty acids are a safe and effective adjunct to atorvastatin, particularly for patients with dyslipidemia characterized by elevated triglycerides.

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