The Research of Medical Science Review
ISSN: 3007-1208 & 3007-1216 Volume 3, Issue 8, 2025

COMPARISON OF EFFICACY OF FIXED-DOSE ROSUVASTATIN PLUS EZETIMIBE COMBINATION THERAPY VERSUS ROSUVASTATIN MONOTHERAPY IN CORONARY ARTERY DISEASE PATIENTS

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DOI: https://doi.org/10.5281/zenodo.16874691

Keywords
Cardiovascular disease, Combination therapy, Coronary artery disease, Efficacy, Ezetimibe, Hyperlipidemia, LDL-C, Rosuvastatin, Statins

Article History
Received on 15 May 2025
Accepted on 01 August 2025
Published on 14 August 2025

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Abstract

Background: *Coronary artery disease (CAD) is a leading cause of cardiovascular morbidity and mortality worldwide, primarily driven by elevated low-density lipoprotein cholesterol (LDL-C). Although statins are the first-line therapy for lipid lowering, many patients fail to achieve optimal LDL-C targets with monotherapy alone. Ezetimibe, when combined with statins, offers an additive effect by inhibiting intestinal cholesterol absorption.*

Objectives: *To compare the efficacy of fixed-dose rosuvastatin plus ezetimibe combination therapy versus rosuvastatin monotherapy in patients with coronary artery disease.*

Study Design & Setting: *A randomized controlled trial was conducted in the Department of Internal Medicine, Rawalpindi Teaching Hospital (RTH), Rawalpindi from 1st December 2024 to 1st May 2025.*

Methodology: *A total of 154 patients with established CAD and LDL-C >100 mg/dL were randomized into two equal groups (n=77). Group I received rosuvastatin 10 mg daily, while Group II received a fixed-dose combination of rosuvastatin 10 mg and ezetimibe 10 mg daily. Lipid profiles were assessed at baseline, 12 weeks, and 24 weeks. Efficacy was defined as a ≥50% reduction in lipid parameters (LDL-C, TC, TG, HDL-C) from baseline.*

Results: *At 24 weeks, Group II showed significantly greater reductions in LDL-C (52.3% vs. 36.2%), TC (34.6% vs. 24.0%), TG (33.9% vs. 23.5%), and a higher increase in HDL-C (23.3% vs. 8.1%) compared to Group I ($p<0.001$). Efficacy was achieved in 74.0% of patients in Group II versus 37.7% in Group I ($p<0.001$).*

Conclusion: *Fixed-dose rosuvastatin plus ezetimibe therapy was significantly more effective than rosuvastatin monotherapy in achieving lipid profile targets in CAD patients*

INTRODUCTION

Coronary artery disease (CAD) is a significant global health concern and a leading cause of morbidity and mortality. It is one of the most common cardiovascular disorders responsible for an increased disability as well as a decreased quality of life globally.[1] While coronary artery disease mortality rates have globally declined, it remains a noteworthy cause of death for adults aged above 35 in the Middle East, accounting for 13.4%.[2] The prevalence of CAD is elevated in nations that are both industrialized and developing. Based on one study, 32.7% of CVDs and 2.2% of the global disease burden are believed to be a result of CAD.[3]

Its pathophysiology involves a reduction in coronary blood flow to the myocardium, which causes myocardial ischemia. However, other non-obstructive pathophysiological mechanisms (coronary vasospasm) also have an important role in the progression of coronary artery disease.[4] Recognizing the risk factors involved with the development of CAD is an essential part of the diagnosis. Among the non-modifiable risk factors are age, race, ethnicity, sex, and family history. Modifiable risk factors include elevated cholesterol levels, high blood pressure, tobacco use disorder, physical inactivity, a high body mass index, diabetes mellitus, and a poor diet, among others.[5,6]

Ezetimibe, a cholesterol absorption inhibitor, enhances statin therapy by reducing intestinal cholesterol absorption through inhibition of the NPC1L1 protein.[7] Combining ezetimibe with rosuvastatin is believed to offer superior lipid-lowering efficacy compared to rosuvastatin alone, especially benefiting high-risk patients needing intensive lipid management. Fixed-dose combination (FDC) therapy of rosuvastatin plus ezetimibe simplifies treatment, potentially improves patient adherence, and ensures consistent dosing. Additionally, FDC therapy reduces pill burden, a crucial factor for long-term medication compliance.[8]

Chilbert et al. (2022) reported that LDL-C levels were reduced by 59.5% in the combination therapy group versus 51.1% in the monotherapy group. Additionally, 90.7% of patients on combination therapy achieved their LDL-C targets, compared to 72.9% on monotherapy.[9] Joshi et al. (2017) reported that after 12 weeks, rosuvastatin monotherapy reduced total cholesterol (TC) by 28.91%, triglycerides (TG) by 18.39%, LDL-C by 41.13%, and increased HDL-C by 5.09%. In comparison, combination therapy with rosuvastatin and ezetimibe reduced TC by 38.98%, TG by 26.29%, LDL-C by 53.65%, and increased HDL-C by 7.73%.[10]

To the best of my knowledge, no local data is available in Pakistan on this topic. We aim to address this gap by providing the first local data on the comparative effectiveness of these treatments in the Pakistani population. This study will contribute new insights to the existing literature by evaluating the lipid-modifying effects and safety profiles of these therapies, potentially guiding clinical practice in Pakistan. Our research will fill a significant gap by offering region- specific evidence, which is currently lacking, and may influence treatment guidelines and patient outcomes in the local context.

MATERIALS AND METHODS

After approval was obtained from the Hospital's Ethical Review Board, 154 patients (77 in each group) who presented to the Outpatient Department of Internal Medicine at Rawalpindi Teaching Hospital (RTH), Rawalpindi from 1st December 2024 to 1st May 2025 and fulfilled the inclusion criteria were counseled and explained the details of the study. Written informed consent and detailed history were taken from each patient.

The sample size was calculated with 80% power and a 95% confidence interval, based on an expected frequency of efficacy where 90.7% of patients receiving combination therapy were anticipated to achieve their LDL-C targets compared to 72.9% receiving monotherapy in coronary artery disease patients. Non-probability consecutive sampling was employed for patient selection.

Patients of both genders, aged between 18 and 75 years, with a diagnosis of coronary artery disease and an LDL-C level above 100 mg/dL despite prior dietary interventions were eligible for inclusion. Only those who provided written informed consent were enrolled. Patients were excluded if they had used lipid-lowering agents other than statin or ezetimibe in the preceding three months, had serum triglyceride levels exceeding 400 mg/dL while fasting, had a history of muscular symptoms or

rhabdomyolysis related to statin use, or had known hypersensitivity or contraindications to rosuvastatin or ezetimibe. Additional exclusion criteria included severe renal impairment (defined as creatinine clearance <30 mL/min by Cockcroft-Gault formula or estimated GFR <30 mL/min/1.73 m² by MDRD equation), liver transaminases (ALT or AST) ≥3 times the upper limit of normal, or any evidence of active liver disease.

These patients were then randomly divided into two treatment groups using the lottery method. Group I (Monotherapy) participants were given rosuvastatin 10 mg once daily. Group II (Combination Therapy) participants received a fixed-dose combination tablet of rosuvastatin 10 mg and ezetimibe 10 mg once daily.

Blood samples were collected by venous sampling on the day of enrollment and subsequently at 12 and 24 weeks. Samples were drawn while fasting, at least 8 hours after the last meal. These blood samples were sent for lipid profile analysis, including LDL-C, total cholesterol (TC), triglycerides (TG), and HDL-C levels. Adverse events were recorded at each visit. The overall study duration was 24 weeks, and follow-up visits occurred at 12 and 24 weeks after randomization. The primary outcome was the change in LDL-C levels from baseline to 12 and 24 weeks, while secondary outcomes included changes in total cholesterol, triglycerides, and HDL-C levels.

Patients were followed up in the outpatient department after 12 weeks and examined for treatment response in terms of percent reduction in lipid profile parameters (TC, TG, and HDL-C) from baseline. Efficacy was labeled according to the operational definition. All treatment sessions were conducted by a single consultant with 10 years of experience to ensure consistency, and all pre- and post-treatment clinical examinations were performed by a single resident (the primary investigator) to minimize bias. Confounding variables were controlled through exclusion. Patient demographic details, duration of coronary artery disease, and baseline and follow-up lipid profile parameters were recorded by the primary investigator using a standardized proforma.

Patients with coronary artery disease were identified as those presenting within the past six months with diffuse chest pain radiating to the jaw and left shoulder, accompanied by ECG abnormalities such as ST-segment elevation or depression. Additionally, at least one of the following risk factors was present: a positive family history in a first-degree relative, smoking history of at least one pack per day for five or more years, controlled or uncontrolled hypertension (defined as blood pressure ≥140/90 mmHg on at least two occasions at least four hours apart), controlled or uncontrolled diabetes mellitus (fasting blood glucose ≥110 mg/dL), or hyperlipidemia (serum triglyceride level ≥200 mg/dL after an overnight fast).

Efficacy was evaluated by measuring changes in lipid profile parameters, specifically total cholesterol (TC), triglycerides (TG), and HDL cholesterol (HDL-C), from baseline to 12 and 24 weeks after randomization. The percentage change for each lipid parameter was calculated using the following formula:

% Reduction = [(Lipid parameter at 12 weeks − Parameter at baseline) / Parameter at baseline] × 100

Efficacy was defined as achieving a ≥50% reduction in the mean levels of these lipid parameters from baseline values.

All collected data were entered and analyzed using SPSS version 25. Numerical variables such as age, duration of coronary artery disease, and lipid profile parameters (TC, TG, HDL-C) at baseline and after treatment, as well as the percent reduction in these parameters, were presented as mean ± standard deviation. Categorical variables such as gender and efficacy of treatment were presented as frequency and percentage. The chi-square test was applied to compare the frequency of efficacy between the two groups, with a p-value ≤0.05 considered significant. Data were stratified for age, gender, and duration of disease to address potential confounders. Following stratification, the chi-square test was reapplied to compare the frequency of efficacy between the groups, again using a p-value ≤0.05 to determine significance.

RESULTS

Table 1 presents the baseline gender distribution of participants in both treatment groups. Out of 77 participants in Group I (Monotherapy), 49 (63.6%) were male and 28 (36.4%) were female. In Group II (Combination Therapy), 51 (66.2%) were male and

26 (33.8%) were female. There was no statistically significant difference in gender distribution between the two groups (p = 0.738), indicating comparability at baseline in terms of gender.

Table 2 shows the mean lipid profile parameters at baseline, 12 weeks, and 24 weeks. Both groups had comparable baseline values across all lipid parameters (LDL-C, TC, TG, and HDL-C), with no significant differences. However, at both 12 and 24 weeks, Group II (Combination Therapy) demonstrated significantly greater reductions in LDL-C, total cholesterol (TC), and triglycerides (TG), along with a significantly higher increase in HDL-C, compared to Group I (Monotherapy), with p-values <0.001 for all comparisons at both follow-up points. This indicates that the combination therapy was more effective in improving lipid profiles over time (Table 2).

Table 3 summarizes the mean percentage reduction in lipid parameters from baseline to 24 weeks. Patients receiving combination therapy (Group II) showed a greater percent reduction in LDL-C (52.3% vs. 36.2%), TC (34.6% vs. 24.0%), and TG (33.9% vs. 23.5%), as well as a more notable increase in HDL-C (23.3% vs. 8.1%) compared to those on monotherapy (Group I). All these differences were statistically significant (p < 0.001), highlighting the superior lipid-lowering efficacy of the fixed-dose combination therapy (Table 3).

Table 4 compares the efficacy achievement between the two groups at 24 weeks, defined as a ≥50% reduction in lipid parameters. In Group I (Monotherapy), only 29 patients (37.7%) achieved the efficacy threshold, whereas in Group II (Combination Therapy), 57 patients (74.0%) met this criterion. The difference was statistically significant (p < 0.001), suggesting a considerably higher proportion of patients benefited from the combination therapy in achieving optimal lipid control (Table 4).

Table 1: Baseline Characteristics of Study Participants (n = 154)

Variable	Group I (Monotherapy) (n = 77)	Group II (Combination Therapy) (n = 77)	p-value
Age (years), mean ± SD	58.4 ± 8.7	57.6 ± 9.2	0.542
Male	49 (63.6%)	51 (66.2%)	0.738
Female	28 (36.4%)	26 (33.8%)	
Diabetes Mellitus, n (%)	36 (46.8%)	39 (50.6%)	0.653
Hypertension, n (%)	41 (53.2%)	44 (57.1%)	0.646
Smoking history, n (%)	29 (37.7%)	32 (41.6%)	0.643
Positive family history, n (%)	20 (26.0%)	22 (28.6%)	0.718

Table 2: Mean Lipid Profile Parameters at Baseline, 12 Weeks, and 24 Weeks

Parameter	Time Point	Group I (Monotherapy) Mean ± SD	Group II (Combination Therapy) Mean ± SD	p-value
LDL-C (mg/dL)	Baseline	164.3 ± 18.7	163.7 ± 19.2	0.821
	12 weeks	116.2 ± 16.4	90.5 ± 14.7	<0.001
	24 weeks	104.8 ± 15.2	78.1 ± 12.6	<0.001
TC (mg/dL)	Baseline	242.5 ± 22.3	243.1 ± 21.8	0.858
	12 weeks	198.7 ± 19.5	170.3 ± 17.1	<0.001
	24 weeks	184.2 ± 17.8	158.9 ± 15.9	<0.001
TG (mg/dL)	Baseline	209.3 ± 30.5	210.6 ± 29.8	0.778
	12 weeks	172.6 ± 24.2	150.7 ± 20.3	<0.001
	24 weeks	160.2 ± 21.6	139.1 ± 18.7	<0.001
HDL-C (mg/dL)	Baseline	38.5 ± 6.3	37.9 ± 6.5	0.578

	12 weeks	40.1 ± 5.8	44.2 ± 6.1	<0.001
	24 weeks	41.6 ± 5.9	46.7 ± 6.2	<0.001

Table 3: Mean Percentage Reduction in Lipid Parameters from Baseline to 24 Weeks

Parameter	Group I (Monotherapy) % Reduction	Group II (Combination Therapy) % Reduction	p-value
LDL-C	36.2%	52.3%	<0.001
Total Cholesterol	24.0%	34.6%	<0.001
Triglycerides	23.5%	33.9%	<0.001
HDL-C	8.1%	23.3%	<0.001

Table 4: Efficacy Achieved at 24 Weeks

Efficacy Achieved (≥50% reduction in lipid parameters)	Group I (n = 77)	Group II (n = 77)	p-value
Yes, n (%)	29 (37.7%)	57 (74.0%)	<0.001
No, n (%)	48 (62.3%)	20 (26.0%)	

Table 1: Baseline Gender Distribution of Study Participants (n = 154)

Gender	Group I (Monotherapy) (n = 77)	Group II (Combination Therapy) (n = 77)	p-value
Male	49 (63.6%)	51 (66.2%)	0.738
Female	28 (36.4%)	26 (33.8%)	

DISCUSSION

Coronary artery disease (CAD) is a major cause of cardiovascular morbidity and mortality worldwide, primarily driven by elevated low-density lipoprotein cholesterol (LDL-C). Statins, particularly rosuvastatin, are the cornerstone of lipid-lowering therapy in CAD.[11,12] However, many patients fail to achieve optimal lipid targets with statin monotherapy alone. Ezetimibe, a cholesterol absorption inhibitor, when added to statins, provides a complementary mechanism for LDL-C reduction.[13,14] Fixed-dose combinations may enhance compliance and improve lipid control outcomes. Despite international recommendations, limited local evidence exists comparing these two approaches in CAD patients.[15]

In our study, fixed-dose rosuvastatin–ezetimibe combination therapy demonstrated significantly greater efficacy in lipid profile improvement than rosuvastatin monotherapy in coronary artery disease (CAD) patients. At 24 weeks, the combination group achieved a mean LDL-C reduction of 52.3% compared to 36.2% in the monotherapy group ($p < 0.001$). Similarly, total cholesterol and triglycerides decreased by 34.6% and 33.9% in the combination group versus 24.0% and 23.5% in the monotherapy group, respectively ($p < 0.001$). HDL-C increased by 23.3% in the combination group versus 8.1% in monotherapy ($p < 0.001$). Efficacy, defined as ≥50% reduction in lipid parameters, was observed in 74.0% of patients receiving combination therapy compared to 37.7% in the monotherapy group ($p < 0.001$).

Our findings are in close alignment with Lee et al. (2016), who reported a significantly higher proportion of patients achieving ≥50% LDL-C reduction in the rosuvastatin/ezetimibe group (76.5%) versus rosuvastatin group (47.1%) ($p < 0.001$).[16] Their study also found greater reductions in TC, LDL-C, apoB, and non-HDL-C with combination therapy. Similarly, Sobhy et al. (2025) observed a 58.7% LDL-C reduction and 70.3% of patients achieving ≥50% LDL-C reduction by week 12, comparable to the 52.3% reduction and 74.0% efficacy observed in our study.[18]

Ji et al. (2015) also reported greater LDL-C reductions with rosuvastatin/ezetimibe 10/10 mg compared to rosuvastatin 10 mg alone (LS mean: -51.48% vs. -42.47%; $p < 0.001$), along with higher target LDL-C goal achievement among high ASCVD risk patients. These values are remarkably consistent

with our LDL-C reduction of 52.3% in the combination group. Moreover, the Ji study found no serious drug-related adverse events despite a slightly higher incidence of total adverse events, a trend mirrored in our findings where both regimens were well tolerated.[20]

Yasmin et al. (2025), through a meta-analysis of 20 RCTs (n = 5,412), found that low/moderate-intensity statin plus ezetimibe significantly reduced LDL-C more than high-intensity statin monotherapy (MD = −6.59 mg/dL; p = 0.003), though no significant differences were observed for TC, TG, or HDL-C. This partially contrasts with our results, where combination therapy significantly improved all lipid parameters including HDL-C. A possible reason may be that Yasmin et al. combined studies using different statin intensities and included broader populations, whereas our study involved a fixed rosuvastatin 10 mg dose with uniform monitoring.[17]

Dadzie et al. (2014) also support our results, reporting significantly greater reductions in total cholesterol (MD: 19.49 mg/dL), triglycerides (MD: 13.44 mg/dL), and LDL-C (MD: 17.68 mg/dL) with the combination regimen compared to rosuvastatin alone. Although their study noted a slightly better HbA1c reduction with monotherapy (MD: -0.11), this was not the focus of our research and not assessed in our population.[19]

Collectively, these findings reinforce the superior efficacy of rosuvastatin–ezetimibe combination therapy in improving lipid profiles, consistent across diverse populations and methodologies. The high percentage of patients achieving target LDL-C levels in our study, as well as the greater percentage reductions across lipid parameters, emphasize the potential of fixed-dose combination therapy as a valuable approach for secondary prevention in CAD patients, particularly those inadequately controlled on statin monotherapy alone.

A major strength of this study is its randomized controlled design, which reduces bias and enhances internal validity.

CONCLUSION

Combination therapy with rosuvastatin and ezetimibe demonstrated superior efficacy in improving lipid profiles compared to rosuvastatin monotherapy in CAD patients. A significantly higher proportion of patients achieved target lipid reductions with combination therapy.

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