

A Non-Interventional, Prospective, Observational Study to Understand the Usage Pattern, Safety and Effect of Rosuvastatin (Roshal™) in Dyslipidemia Patients from Democratic Republic of Congo (PEARL Study)

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Abstract

Background: Dyslipidemia is a major modifiable risk factor for cardiovascular disease, which remains a leading cause of morbidity and mortality globally. In sub-Saharan Africa, including the Democratic Republic of Congo (DRC), the prevalence of dyslipidemia is substantial and rising due to urbanization and changing lifestyles. Rosuvastatin is among the most potent statins available, but real-world data on its usage patterns, safety, and effectiveness in the Congolese population remain limited. **Methods:** This was a prospective, multicenter, non-interventional, observational study conducted across multiple clinical sites in the Democratic Republic of Congo. Adult patients (≥ 18 years) with dyslipidemia prescribed rosuvastatin (Roshal™) as part of routine clinical care were enrolled. Data on demographics, clinical characteristics, lipid parameters, treatment adherence, and adverse events were collected at baseline (visit 1) and at visit 2 (week 12). Changes in lipid parameters and blood pressure were assessed using paired t-tests. **Results:** A total of 381 patients were enrolled (54.6% female; mean age 56.38 ± 13.20 years). The most common comorbidities were hypertension (58.8%), diabetes (40.2%), and obesity (2.1%). Most patients (50.1%) were prescribed rosuvastatin 10 mg (Roshal™); 28.3% received 20 mg; and 21.3% received 5 mg. Rosuvastatin (Roshal™) was newly initiated in all patients; 5.2% had previously received another lipid-lowering medication before switching, most commonly simvastatin. Rosuvastatin significantly reduced LDL-C (18.75% reduction, $p = 0.001$), total cholesterol (15.70% reduction, $p < 0.001$), and triglycerides (15.64% reduction, $p < 0.001$).

from baseline to week 12. Unadjusted within-group LDL-C reductions varied by dose, from 9.22% (5 mg) to 25.29% (10 mg) and 18.81% (20 mg) ($p \leq 0.023$ for all), without a consistent dose-response pattern. Systolic and diastolic blood pressure also showed significant reductions (12.59% and 11.91%, respectively; $p < 0.001$ for both). Adherence was moderate to high, with 57.52% of patients showing medium adherence and 37.58% showing high adherence at week 12. Adverse events were reported in only 2.8% of patients, all of mild to moderate severity. Physicians rated the global efficacy and safety as excellent or good in 75.76% of cases. **Conclusion:** Rosuvastatin (Roshal™) demonstrated favorable efficacy and safety profiles in Congolese patients with dyslipidemia in real-world clinical practice, with significant improvements in lipid parameters and blood pressure and a low incidence of adverse events. These findings support the use of rosuvastatin as an effective and well-tolerated option for dyslipidemia management in the Congolese population.

Keywords

Rosuvastatin, Dyslipidemia, Democratic Republic of Congo, Real-World Evidence, Observational Study, Lipid Profile, Safety, Adherence

1. Introduction

Cardiovascular disease remains the leading cause of mortality worldwide and contributes substantially to the rising non-communicable disease burden in sub-Saharan Africa (SSA), where it now co-exists with the long-standing burden of communicable, maternal, and nutritional disorders [1]. Although atherosclerosis-driven ischaemic heart disease historically appeared less common in SSA than in high-income countries, the Global Burden of Diseases 2010 analysis highlighted that CVDs in this region are uniquely distributed—about half are non-atherosclerotic—and that the deaths from CVD occur at substantially younger ages than elsewhere in the world [2]. A ten-year hospital survey in Kinshasa reported that CVD accounted for 31% of medical admissions and 35% of in-hospital deaths, with stroke being the predominant contributor to cardiovascular mortality [3]. In a national-level analysis covering the DRC, the prevalence of hypertension rose rapidly over a five-year period (15.1%) and CVDs and diabetes respectively accounted for 20.4% and 5.4% of mortality, yet health facilities were generally not prepared to manage these conditions [4]. These signals indicate a pressing need for evidence-based lipid management strategies tailored to Central African populations.

Dyslipidemia is a well-established, causally related driver of atherosclerotic cardiovascular disease. The 2019 European Society of Cardiology and European Atherosclerosis Society guidelines eliminated the older “LDL-C hypothesis” in favour of an established causal relationship, lowering LDL-C goals to <55 mg/dL for very high-risk patients, <70 mg/dL for high-risk patients, and <100 mg/dL for moderate-risk patients [5] [6]. Pharmacological management in these guidelines recom-

mends high-intensity statins up to the highest tolerated dose as first-line therapy, with ezetimibe and, where needed, PCSK9 inhibitors as add-on agents when targets are not achieved [7]-[9]. Despite the strength of these recommendations, international observational data show that LDL-C goal attainment remains poor: in the International Cholesterol Management Practice Study covering 18 countries outside Western Europe, including several in Africa and the Middle East, only 32.1% of very high-risk patients on stable lipid-lowering therapy achieved their LDL-C target [10] [11].

Among the statins, rosuvastatin offers favourable pharmacological characteristics: high affinity for HMG-CoA reductase due to additional binding interactions, relative hydrophilicity, selective hepatic uptake, low CYP3A4 metabolism, and a long half-life [12]-[14]. Rosuvastatin 10 - 40 mg reduces LDL-C by 52% - 63%, increases HDL-C by up to 14%, and reduces triglycerides by up to 28%, with 10% - 20% additional LDL-C reduction compared with atorvastatin across equivalent dose ranges [12] [13] [15]. Landmark trials such as JUPITER established that rosuvastatin 20 mg reduced major cardiovascular events by 44% (HR 0.56, 95% CI 0.46 - 0.69) and all-cause mortality by 20% (HR 0.80, 95% CI 0.67 - 0.97) in apparently healthy individuals with elevated high-sensitivity C-reactive protein [16] [17]. Subsequent real-world studies from Asia, including a multicentre Indian study of rosuvastatin plus ezetimibe (mean LDL-C reductions of 92 - 100 mg/dL across statin-intensity strata) and a retrospective Nepalese study (mean LDL-C reduction of 63.4 mg/dL with rosuvastatin 10 mg, with only 3.03% adverse events), have confirmed consistent efficacy and tolerability in routine practice [18] [19].

However, SSA populations remain under-represented in such evidence. Because of pharmacogenomic differences in transporters and metabolising enzymes (ABCG2, SLCO1B1, CYP2C9, APOE, and HMG-CoR) between African and Caucasian or Asian populations, the efficacy, dose-response relationship, and safety profile of rosuvastatin cannot always be extrapolated from non-African trials [20]. Real-world evidence from the Democratic Republic of the Congo is therefore essential to inform local treatment decisions, identify barriers to adherence, and contextualise international guideline recommendations.

The PEARL study was designed to address this gap. Its primary objective was to document the real-world usage pattern of rosuvastatin in DRC patients with dyslipidemia. Secondary objectives were to quantify within-patient changes in lipid parameters, assess anthropometric and blood-pressure trends, describe treatment adherence and persistence, and catalogue adverse events.

2. Methods

2.1. Study Design and Setting

The PEARL study was a prospective, multicenter, non-interventional, observational study conducted in routine clinical practice in the DRC. The study was designed to mirror real-life prescribing, with no protocol-mandated interventions beyond standard-of-care clinician decisions. The study was conducted across ap-

proximately 15 participating sites, comprising general hospitals and private outpatient clinics, with patient recruitment occurring between December 2024 and January 2025. At each site, consecutive eligible patients presenting during the recruitment period were enrolled in accordance with the inclusion and exclusion criteria defined in the study protocol. Each participating investigator documented patient characteristics, dispensed rosuvastatin doses, baseline lipid values, anthropometric measurements, blood-pressure readings, adherence behaviour, and any adverse events at the baseline visit and at the first scheduled follow-up.

2.2. Study Population

The target population comprised adult patients with dyslipidemia who were clinically suitable for rosuvastatin prescription in the real-world setting.

2.3. Inclusion Criteria

- 1) Female or male patients aged 18 years or above.
- 2) Patients with documented dyslipidemia who were clinically suitable for the prescription of rosuvastatin according to the locally approved prescribing information.
- 3) Patients willing to provide informed consent for the use of their personal and health data prior to study entry.

2.4. Exclusion Criteria

- 1) Presence of any contraindication to rosuvastatin as specified in the locally approved prescribing information.
- 2) History of statin-induced myopathy or serious hypersensitivity reaction to any HMG-CoA reductase inhibitor, including rosuvastatin.
- 3) Any medical or non-medical condition that, in the opinion of the treating physician, could prevent the patient's participation in the study.
- 4) Pregnant or lactating women, and women of childbearing potential unwilling to use an effective barrier contraceptive method for the duration of the study.

2.5. Sample Size

A total of 381 adult patients presenting at participating outpatient centres were enrolled at the baseline (visit 1) visit and followed at 3 months $\pm$ 7 days (visit 2).

2.6. Demographic and Clinical Data

Each participating investigator documented patient characteristics, dispensed rosuvastatin doses, baseline lipid values, anthropometric measurements, blood-pressure readings, adherence behaviour, and any adverse events at the baseline visit and at visit 2.

2.7. Study Objectives and Endpoints

The primary endpoint was the within-patient percent change in LDL-C from

baseline to visit 2, stratified by rosuvastatin dose. Secondary endpoints included total cholesterol, HDL-C, triglycerides, weight, systolic blood pressure and diastolic blood pressure, adherence (categorised as poor, medium, or high), and adverse-event frequency and severity (mild, moderate, severe).

2.8. Statistical Analysis

Continuous variables were summarised as mean $\pm$ standard deviation; pre-/post-comparisons were performed using paired t-tests. Categorical variables were expressed as frequency and percentage. All analyses were performed on available-case data. A p-value < 0.05 was considered statistically significant.

2.9. Ethical Considerations

Because PEARL was non-interventional and used routine clinical data, informed consent was obtained according to local regulations and the study was conducted in accordance with applicable national guidelines and the principles of the Declaration of Helsinki.

3. Results

3.1. Patient Disposition and Baseline Characteristics

A total of 381 patients were enrolled at baseline. The mean age of the participants was 56.38 ± 13.20 years. The majority of participants were in the age group of 51 - 60 years (27.3%), followed by the age group 61 - 70 years (24.9%). Females comprised 54.6% of the sample (208 participants), while males represented 45.4% (173 participants), as shown in **Table 1**.

Table 1. Baseline demographic and clinical characteristics.

Characteristic	N (%)
Total Patients	381 (100%)
Sex	
Female	208 (54.6%)
Male	173 (45.4%)
Age Group	
18 - 20 years	3 (0.8%)
21 - 30 years	9 (2.4%)
31 - 40 years	37 (9.7%)
41 - 50 years	77 (20.2%)
51 - 60 years	104 (27.3%)
61 - 70 years	95 (24.9%)
71 - 80 years	48 (12.6%)
Above 80 years	8 (2.1%)

Continued

Risk Factors	
Tobacco use	96 (25.2%)
Alcohol consumption	181 (47.5%)
Physical inactivity	140 (36.7%)
Poor dietary habits	190 (49.9%)

The most prevalent risk factors were poor dietary habits (49.9%) and alcohol consumption (47.5%), followed by physical inactivity (36.7%) and tobacco use (25.2%). The high prevalence of these modifiable risk factors highlights the need for comprehensive lifestyle interventions in this population, as shown in **Table 2**.

Table 2. Medical history and comorbidities.

Characteristic	N (%)
Duration of Dyslipidemia	
Newly diagnosed	93 (44.9%)
6 months - 1 year	100 (48.3%)
1 - 3 years	14 (6.8%)
Family History of Dyslipidemia	
Yes	137 (36.4%)
No	239 (63.6%)
Comorbidities	
Hypertension	224 (58.8%)
Diabetes	153 (40.2%)
Obesity	8 (2.1%)
Heart disease	8 (2.1%)

The majority of patients (44.9%) were newly diagnosed with dyslipidemia, indicating a high recent increase in diagnosis. Hypertension was the most common comorbidity (58.8%), followed by diabetes (40.2%). The high prevalence of hypertension and diabetes is consistent with the clustering of cardiovascular risk factors in this population. A family history of dyslipidemia was present in 36.4% of patients.

3.2. Rosuvastatin (Roshal™) Usage Pattern

Rosuvastatin (Roshal™) was newly initiated in all 381 enrolled patients; it was not added on top of ongoing statin therapy in any patient. Dose at initiation was documented for 371 of the 381 patients (10 patients had missing dose data); among these, the most frequently prescribed dose was 10 mg (50.1%, n = 186), followed by 20 mg (28.3%, n = 105), 5 mg (21.3%, n = 79), and 40 mg (0.3%, n = 1). Prior

lipid-lowering treatment status was documented for 368 of the 381 patients (13 patients had missing data); of these, the overwhelming majority of patients (94.8%, $n = 349$) had not received any prior lipid-lowering medication, while 5.2% ($n = 19$) had previously been treated with another lipid-lowering agent before switching to rosuvastatin. Among these 19 switched patients, the prior medication was most commonly simvastatin (57.9%), followed by fenofibrate (26.3%) and atorvastatin (15.8%); the most frequently cited reasons for switching were high cost of the prior therapy, poor efficacy, poor compliance, and poor tolerability/safety (**Figure 1**).

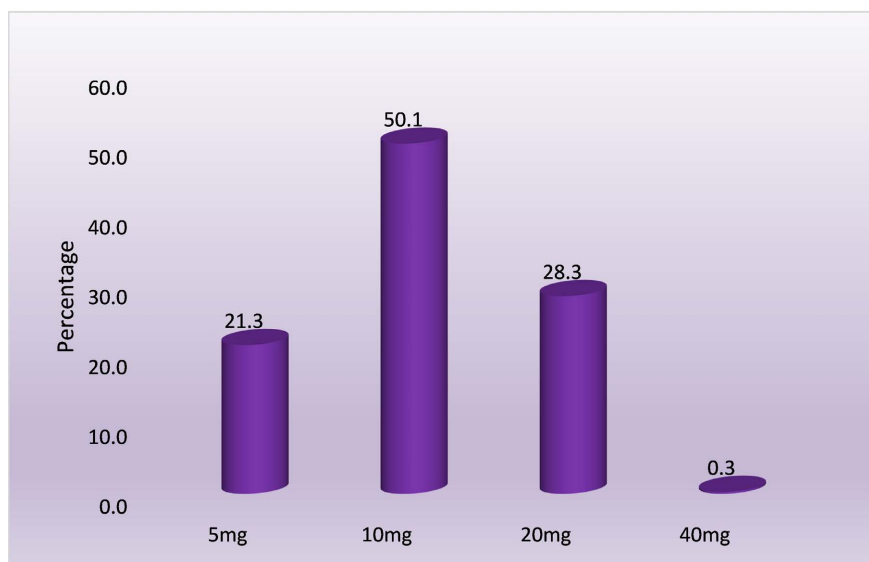


Figure 1. Rosuvastatin dose distribution at baseline (%).

3.3. Switching from Other Lipid-Lowering Medications

Because rosuvastatin was newly initiated in every patient, baseline lipid values for the 349 patients (94.8%) with no prior lipid-lowering therapy can be interpreted as treatment-naïve. For the 19 patients (5.2%) who switched from another lipid-lowering agent, however, baseline values were measured while the prior therapy was still in effect and therefore reflect a treated, rather than an untreated, baseline; this should be considered when interpreting the overall baseline lipid values. Among these 19 patients, the most common prior medication was simvastatin (57.9%), followed by fenofibrate (26.3%) and atorvastatin (15.8%).

3.4. Changes in Lipid Parameters

Significant improvements were observed in all lipid parameters from baseline to week 12.

Of the 381 patients enrolled at baseline, 368 (96.6%) attended the week 12 visit and 13 (3.4%) were lost to follow-up. As PEARL was conducted in a real-world, uncontrolled setting, some asymptomatic patients did not return for their scheduled follow-up visit, and a modest natural attrition rate was therefore expected.

Because PEARL used available-case analysis, the number of patients with paired baseline and week-12 measurements varied further by parameter (Table 3), reflecting which patients had both baseline and week-12 values recorded for that particular measurement.

Table 3. Changes in lipid parameters from baseline to week 12.

Parameter	Baseline (Mean)	Week 12 (Mean)	Mean % Change	p-value	N (paired)
LDL-C (mg/dL)	158.01	128.37	-18.75%	0.001	135
HDL-C (mg/dL)	56.58	54.83	-3.09%	0.374	138
Total Cholesterol (mg/dL)	211.53	178.33	-15.70%	<0.001	163
Triglycerides (mg/dL)	137.95	116.38	-15.64%	<0.001	132

LDL-C: LDL-C levels decreased significantly from baseline to week 12, with a mean reduction of 18.75% ($p = 0.001$). Unadjusted within-group changes by dose were: 5 mg (-9.22%, $p = 0.006$), 10 mg (-25.29%, $p = 0.023$), and 20 mg (-18.81%, $p = 0.002$). As the reduction at 20 mg was smaller than at 10 mg, these dose-specific findings do not indicate a clear dose-response relationship and are more likely to reflect confounding by baseline risk, baseline LDL-C, and prescribing indication than a true pharmacodynamic gradient.

HDL-C: HDL-C levels showed a non-significant decrease of 3.09% from baseline to week 12 ($p = 0.374$). The dose-wise analysis showed no significant changes across any dose group: 5 mg (-3.60%, $p = 0.444$), 10 mg (-1.51%, $p = 0.797$), and 20 mg (-5.33%, $p = 0.359$).

Total Cholesterol: Total cholesterol decreased significantly from baseline to week 12, with a mean reduction of 15.70% ($p < 0.001$). The dose-wise analysis showed significant reductions in the 10 mg (-36.20%, $p < 0.001$) and 20 mg (-41.26%, $p < 0.001$) groups.

Triglycerides: Triglycerides decreased significantly from baseline to week 12, with a mean reduction of 15.64% ($p < 0.001$). The dose-wise analysis showed significant reductions in the 10 mg (-25.51%, $p < 0.001$) and 20 mg (-35.22%, $p < 0.001$) groups.

Table 4. Anthropometric and blood-pressure changes.

	Baseline (Mean)	Week 12 (Mean)	Mean Change	N (paired)
Weight	79.96 kg	76.66 kg	-3.3 kg	320
SBP	153.65 mmHg	134.30 mmHg	-19.35 mmHg	311
DBP	90.13 mmHg	79.40 mmHg	-10.73 mmHg	311

Systolic blood pressure decreased significantly from 153.65 mmHg at baseline to 134.30 mmHg at week 12 (mean reduction: 12.59%, $p < 0.001$). Diastolic blood pressure decreased significantly from 90.13 mmHg at baseline to 79.40 mmHg at

week 12 (mean reduction: 11.91%, $p < 0.001$), see [Table 4](#).

3.5. Patient Adherence

At visit 2 (week 12), 37.58% of patients showed high adherence, 57.52% showed medium adherence, and 4.90% showed poor adherence to rosuvastatin therapy. The moderate to high adherence rates observed are encouraging and suggest that patients were generally compliant with their medication regimen ([Figure 2](#)).

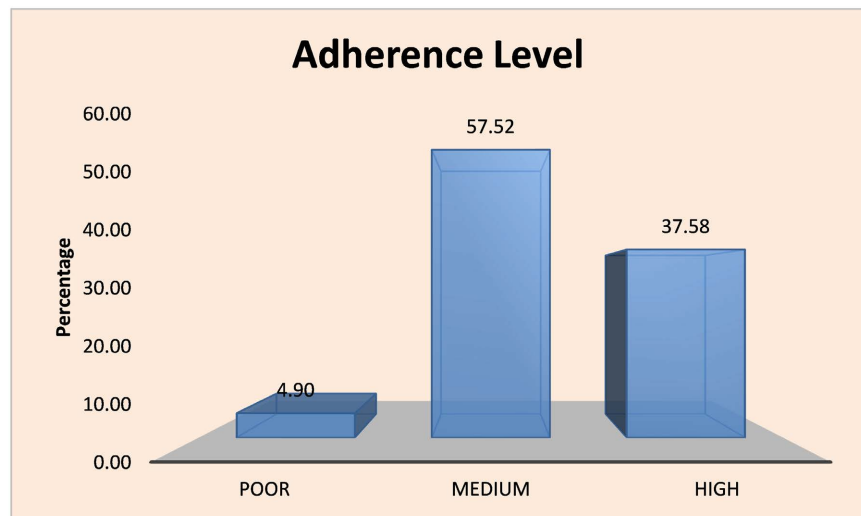


Figure 2. Adherence level.

3.6. Adverse Events

Adverse events were reported in only 2.8% of patients (11 out of 381 patients). The reported adverse events included: dizziness (2), diarrhea (2), physical asthenia (2), dyspepsia (1), headache (1), stomach aches (1), temporary itching (1), and tiredness (1). Of the 11 cases, 9 were of mild severity and 2 were of moderate severity. In 4 cases, the dose of study medication was reduced, and in the remaining 7 cases, no action was taken. No serious adverse events were reported.

3.7. Global Assessment of Efficacy and Safety by Physicians

Overall, 75.76% of physicians rated the efficacy and safety as excellent or good, indicating a high level of satisfaction with rosuvastatin therapy in this patient population ([Figure 3](#)).

4. Discussion

The PEARL study contributes one of the first multicentre, real-world evidence datasets on rosuvastatin use in the DRC, addressing a critical evidence gap given the rising cardiovascular and metabolic disease burden in Central Africa [1] [3]. The demographic profile of the cohort—predominantly middle-aged, with a female majority and a high prevalence of cardiometabolic risk factors—mirrors the broader epidemiological transition described in DRC mortality data, where cardi-

ovascular and metabolic causes together accounted for roughly a quarter of all deaths even within health facilities not optimally equipped for NCD care [3].

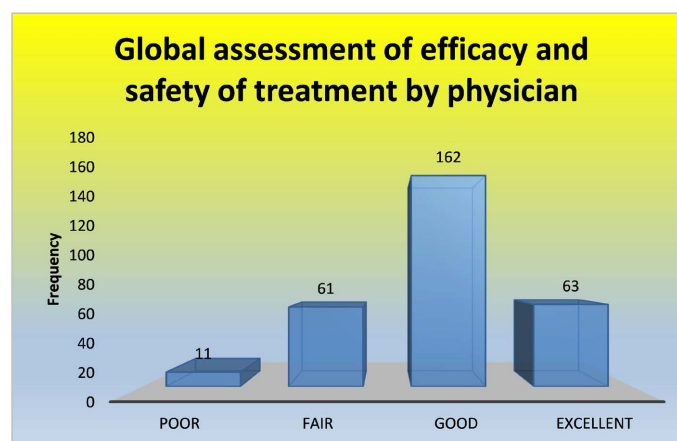


Figure 3. Global assessment of efficacy and safety of treatment by physician.

The headline finding of PEARL is that rosuvastatin (Roshal™), prescribed at predominantly moderate (10 mg) intensities, achieved clinically and statistically meaningful reductions in LDL-C, total cholesterol, and triglycerides within a single follow-up cycle. The overall LDL-C reductions by dose (−9.22% at 5 mg, −25.29% at 10 mg, and −18.81% at 20 mg) align with the pharmacological profile of rosuvastatin reported in dose-ranging and comparator studies, in which 10 - 40 mg of rosuvastatin lowers LDL-C by roughly 50% - 63%—here attenuated because of the relatively short follow-up window and the inclusion of dose groups with small sample sizes [12]-[15]. The pattern across dose groups is broadly consistent with reviews noting that rosuvastatin allows greater proportions of patients to attain LDL-C targets earlier than other statins, reducing the need for up-titration [12] [14]. The total-cholesterol reduction of 15.70% and the triglyceride reduction of 15.64% further confirm the pleiotropic action of rosuvastatin on multiple lipid fractions, mirroring the JUPITER trial—in which rosuvastatin 20 mg reduced LDL-C by 50%, hs-CRP by 37%, and triglycerides by 17% within 12 months—and the consistent efficacy observed in retrospective multicenter studies, such as a 63.4 mg/dL mean LDL-C reduction with rosuvastatin 10 mg in a Nepalese cohort (95% CI 43.0 - 83.8, $p < 0.001$) [16] [17] [19].

The PEARL efficacy findings concur with those reported in landmark trials and intensive-care cohorts. In head-to-head real-world comparisons in Bangladesh, rosuvastatin 5 mg produced a larger LDL-C reduction than atorvastatin 10 mg (135.6 → 95.3 mg/dL vs. 140.7 → 110.4 mg/dL, $p = 0.027$) with fewer adverse events [21]. An Indian multicentre study of rosuvastatin plus ezetimibe in statin-pre-treated patients reported mean LDL-C reductions of 92 - 100 mg/dL across low-, moderate-, and high-intensity baseline statin strata [18]. The within-dose reductions in the PEARL study were statistically significant for LDL-C at every dose (5 mg $p = 0.006$; 10 mg $p = 0.023$; 20 mg $p = 0.002$), confirming a real within-group

lipid-lowering effect at each dose even in this lower-resource setting. However, because the reduction observed at 20 mg (−18.81%) was smaller than that at 10 mg (−25.29%), these unadjusted within-group comparisons should not be interpreted as evidence of a dose-response relationship; they more plausibly reflect confounding by baseline risk, baseline LDL-C, and prescribing indication (e.g., higher doses being preferentially prescribed to patients with more severe or difficult-to-control dyslipidemia).

The HDL-C and triglyceride patterns observed in PEARL are also coherent with international evidence. The triglyceride reduction of 15.64% overall, and the dose-stratified reductions of −25.51 mg/dL (10 mg) and −35.22 mg/dL (20 mg), fall within the 10% - 28% range described for rosuvastatin [13] [14]. This is clinically meaningful in a Central African cohort in which mixed dyslipidemia and metabolic syndrome are increasingly common and in which triglyceride-rich lipoprotein remnants are emerging as additional targets of therapy [7].

The mean reductions observed in systolic (−19.35 mmHg) and diastolic (−10.73 mmHg) blood pressure likely reflect concomitant antihypertensive therapy rather than a direct statin effect—although statin-induced improvements in endothelial function may contribute a small independent component, as suggested in reviews of statin pleiotropy [15]. The high proportion of patients achieving medium (57.52%) or high (37.58%) adherence, with only 4.90% classified as poor, indicates that rosuvastatin was well accepted and integrated into the daily routine of this DRC cohort. This is encouraging given international data showing that long-term adherence to lipid-lowering therapy declines over months and years, with worse persistence typically observed among women and among patients on multiple agents in real-world German and Australian cohorts [22] [23]. In sub-Saharan Africa, undertreatment remains the larger problem: ACE sub-analysis showed that more than three-quarters of eligible outpatients in the region are not on any statin therapy, including two-thirds of those with prior coronary artery disease, cerebrovascular disease, peripheral arterial disease or diabetes [11]. The relatively high adherence observed in PEARL may be attributable to once-daily rosuvastatin dosing (no major CYP3A4 drug-to-drug interactions, allowing safe co-prescription with antihypertensives and antidiabetics) and to the simplicity of long-term adherence messaging in the participating centres.

The overall AE rate of 2.8% in PEARL is consistent with—and slightly lower than—rates reported elsewhere. In the Korean ROSulord® study, rosuvastatin showed favourable safety with infrequent adverse drug reactions in real-world use [24]; a retrospective multicentre study from Nepal reported AEs in only 3.03% of patients [19]; and the HILL-India rosuvastatin-ezetimibe combination study described rosuvastatin-based therapy as well tolerated across dose strata [18]. The PEARL safety profile—predominantly mild, with no severe AEs and no AE-related deaths—is consistent with the safety profile established for rosuvastatin in larger phase III/IV programmes [12] [13] [17].

The PEARL study has several limitations that should be considered when inter-

preting the findings. First, this was a single-country study conducted in the Democratic Republic of Congo, which may limit the generalizability of the findings to other African populations with different demographic profiles, healthcare systems, and genetic backgrounds. The non-interventional, observational design with no control group limits the ability to establish causality or to compare rosuvastatin with other lipid-lowering therapies or placebo. The absence of long-term outcome data, such as major adverse cardiovascular events, hospitalizations, or mortality, precludes assessment of the clinical impact of the observed lipid improvements.

5. Conclusion

In this prospective, multicentre, real-world PEARL cohort from the Democratic Republic of the Congo, rosuvastatin (Roshal™)—predominantly prescribed at 10 mg—produced clinically and statistically significant reductions in LDL-C, total cholesterol, and triglycerides across all dose groups, accompanied by modest improvements in blood pressure and weight. The drug was well tolerated: only 2.8% of patients experienced an adverse event, none of which were severe, and adherence was predominantly medium-to-high. These findings support the integration of rosuvastatin into routine CVD prevention pathways in the DRC and provide locally relevant evidence that complements international trial data and guideline recommendations.

Author Contributions

Dr. Tresor Mude & Dr. Raymond Dinaouz conceived and designed the study. Dr. Tresor Mude & Dr. Raymond Dinaouz collected and analyzed the data. Dr. Tresor Mude & Dr. Raymond Dinaouz prepared the original manuscript draft. Dr. Tresor Mude & Dr. Raymond Dinaouz critically reviewed and edited the manuscript. Dr. Tresor Mude & Dr. Raymond Dinaouz supervised the study. All authors reviewed and approved the final manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Yuyun, M.F., Sliwa, K., Kengne, A.P., Mocumbi, A.O. and Bukhman, G. (2020) Cardiovascular Diseases in Sub-Saharan Africa Compared to High-Income Countries: An Epidemiological Perspective. *Global Heart*, **15**, Article No. 15. <https://doi.org/10.5334/gh.403>
- [2] Moran, A., Forouzanfar, M., Sampson, U., Chugh, S., Feigin, V. and Mensah, G. (2013) The Epidemiology of Cardiovascular Diseases in Sub-Saharan Africa: The Global Burden of Diseases, Injuries and Risk Factors 2010 Study. *Progress in Cardiovascular Diseases*, **56**, 234-239. <https://doi.org/10.1016/j.pcad.2013.09.019>
- [3] Bakilo, E.L. (2024) Cardiovascular Morbidity and Mortality in Sub-Saharan Africa; Wide Hospital Survey in Democratic Republic of Congo. *Journal of Quality in Health Care & Economics*, **7**, 1-8. <https://doi.org/10.23880/jqhe-16000401>

- [4] Angendu Baki, K. (2025) Epidémiologie des Maladies Cardiovasculaires et Métaboliques en République Démocratique du Congo. Doctorat ès Biologie Chimie Santé mention Santé publique, épidémiologie, environnement et sociétés. <https://theses.fr/2025LIMO0066>
- [5] Descamps, O.S., Verhaegen, A., Demeure, F., Langlois, M., Rietzschel, E., Mertens, A., *et al.* (2020) Evolving Concepts on the Management of Dyslipidaemia. *Acta Clinica Belgica*, **75**, 80-90. <https://doi.org/10.1080/17843286.2019.1702823>
- [6] Mach, F., Baigent, C., Catapano, A.L., Koskinas, K.C., Casula, M., Badimon, L., *et al.* (2020) 2019 ESC/EAS Guidelines for the Management of Dyslipidaemias: Lipid Modification to Reduce Cardiovascular Risk. *European Heart Journal*, **41**, 111-188.
- [7] Pirillo, A., Casula, M. and Catapano, A.L. (2023) European Guidelines for the Treatment of Dyslipidaemias: New Concepts and Future Challenges. *Pharmacological Research*, **196**, Article ID: 106936. <https://doi.org/10.1016/j.phrs.2023.106936>
- [8] Sakuma, M., Iimuro, S., Shinozaki, T., Kimura, T., Nakagawa, Y., Ozaki, Y., *et al.* (2022) Optimal Target of LDL Cholesterol Level for Statin Treatment: Challenges to Monotonic Relationship with Cardiovascular Events. *BMC Medicine*, **20**, Article No. 441. <https://doi.org/10.1186/s12916-022-02633-5>
- [9] Rossi, M., Fabris, E., Barbisan, D., Massa, L. and Sinagra, G. (2022) Lipid-Lowering Drug Therapy: Critical Approach for Implementation in Clinical Practice. *American Journal of Cardiovascular Drugs*, **22**, 141-155. <https://doi.org/10.1007/s40256-021-00497-3>
- [10] Danchin, N., Almahmeed, W., Al-Rasadi, K., Azuri, J., Berrah, A., Cuneo, C.A., *et al.* (2018) Achievement of Low-Density Lipoprotein Cholesterol Goals in 18 Countries Outside Western Europe: The International Cholesterol Management Practice Study (ICLPS). *European Journal of Preventive Cardiology*, **25**, 1087-1094. <https://doi.org/10.1177/2047487318777079>
- [11] Hamoui, O., Omar, M.I., Raal, F.J., Rashed, W., Kane, A., Alami, M., *et al.* (2019) Increases in Statin Eligibility to Reduce Cardiovascular Risk According to the 2013 ACC/AHA Cholesterol Guidelines in the Africa Middle East Region: A Sub-Analysis of the Africa Middle East Cardiovascular Epidemiological (ACE) Study. *BMC Cardiovascular Disorders*, **19**, Article No. 61. <https://doi.org/10.1186/s12872-019-1034-2>
- [12] Olsson, A.G., McTaggart, F. and Raza, A. (2002) Rosuvastatin: A Highly Effective New HMG-CoA Reductase Inhibitor. *Cardiovascular Drug Reviews*, **20**, 303-328. <https://doi.org/10.1111/j.1527-3466.2002.tb00099.x>
- [13] Rosenson, R.S. (2003) Rosuvastatin: A New Inhibitor of HMG-CoA Reductase for the Treatment of Dyslipidemia. *Expert Review of Cardiovascular Therapy*, **1**, 495-505. <https://doi.org/10.1586/14779072.1.4.495>
- [14] White, C.M. (2002) A Review of the Pharmacologic and Pharmacokinetic Aspects of Rosuvastatin. *Journal of Clinical Pharmacology*, **42**, 963-970. <https://doi.org/10.1177/009127002401102876>
- [15] Luvai, A., Mbagaya, W., Hall, A.S. and Barth, J.H. (2012) Rosuvastatin: A Review of the Pharmacology and Clinical Effectiveness in Cardiovascular Disease. *Clinical Medicine Insights: Cardiology*, **6**, 17-33. <https://doi.org/10.4137/cmc.s4324>
- [16] Kones, R. (2010) Rosuvastatin, Inflammation, C-Reactive Protein, JUPITER, and Primary Prevention of Cardiovascular Disease—A Perspective. *Drug Design, Development and Therapy*, **4**, 383-413. <https://doi.org/10.2147/dddt.s10812>
- [17] Ridker, P.M., Danielson, E., Fonseca, F.A.H., Genest, J., Gotto, A.M., Kastelein, J.J.P., *et al.* (2008) Rosuvastatin to Prevent Vascular Events in Men and Women with Elevated C-Reactive Protein. *New England Journal of Medicine*, **359**, 2195-2207.

- <https://doi.org/10.1056/nejmoa0807646>
- [18] Nagarajan, R., Nitthiyan, P., Guthe, A.A., Naigude, S.D., Rajesh, M., Kannan, P., *et al.* (2026) High-Intensity Lipid-Lowering Therapy with Rosuvastatin and Ezetimibe for Uncontrolled Dyslipidemia in Indian Patients: A Real-World Study (HILL-INDIA Study). *Heart India*, **14**, 45-53.
https://doi.org/10.4103/heartindia.heartindia_21_25
 - [19] Shakya, S., Shah, D., Kurumbang, J., Sah, A., Mahule, A. and Pednekar, A. (2024) Clinical Experience of Rosuvastatin in Patients with Cardiovascular Events: Insights from a Real-World Study. *Global Journal for Research Analysis*, **13**, 41-45.
<https://doi.org/10.36106/gjra/8001602>
 - [20] Soko, N.D., Masimirembwa, C. and Dandara, C. (2016) Pharmacogenomics of Rosuvastatin: A Glocal (Global + Local) African Perspective and Expert Review on a Statin Drug. *OMICS: A Journal of Integrative Biology*, **20**, 498-509.
<https://doi.org/10.1089/omi.2016.0114>
 - [21] Hossain, M.B., Amin, R. and Jalal Uddin, M. (2024) Assessment of Efficacy and Adverse Effects of Atorvastatin and Rosuvastatin in Dyslipidemia Treatment in a Tertiary Care Hospital. *TAF: Journal of Teachers Association*, **37**, 833-840.
<https://doi.org/10.70818/taj.v037i02.0552>
 - [22] Rakhshanda, S., Rye, K.A., Liaw, S.T., Rhee, J. and Jonnagaddala, J. (2026) Predictors of Statin Adherence in Primary Care Using Real-World Data.
<https://www.medrxiv.org/content/10.64898/2026.02.24.26347032v2>
 - [23] Koenig, W., Lorenz, E.S., Beier, L. and Gouni-Berthold, I. (2024) Retrospective Real-World Analysis of Adherence and Persistence to Lipid-Lowering Therapy in Germany. *Clinical Research in Cardiology*, **113**, 812-821.
<https://doi.org/10.1007/s00392-023-02257-6>
 - [24] Kim, D.Y., Kim, S.H., Kim, E., Han, S., Park, J., Youn, J., *et al.* (2025) ROsulord® Safety for Patients with Dyslipidemia Study: A Non-Interventional, Multicenter, Prospective, Observational Study in South Korea. *Cardiology and Therapy*, **14**, 17-29.
<https://doi.org/10.1007/s40119-024-00391-4>