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Original Article

Efficacy of Statin and Non-Statin Therapies for Lipid Abnormalities in CKD: Evidence from Ayub Teaching Hospital

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A B S T R A C T

Objective: To compare the effectiveness and safety of statin therapy with non-statin management in improving lipid profile and renal outcomes among patients with chronic kidney disease (CKD).

Methodology: The study is a prospective cohort study that will be carried out at the Department of Nephrology, Ayub Teaching Hospital (ATH), Abbottabad, Pakistan, during the period of January to December 2024. Statin therapy (n = 75) and non-statin management (n = 75) comprised the two groups of 150 CKD patients in this comparative analysis. Age, gender, body mass index (BMI), comorbidities, stage of chronic kidney disease (CKD), estimated glomerular filtration rate (eGFR), and lipid profile were among the baseline clinical and demographic features that were documented. Patients were followed for six months. The primary outcomes were achievement of LDL-C <70 mg/dL or ≥50% reduction in LDL-C and mean reduction in LDL-C levels. Secondary outcomes included changes in non-HDL cholesterol, triglycerides, HDL-C, and renal function (eGFR). Safety outcomes such as adverse events, myalgia, elevation of liver enzymes, and major adverse cardiovascular events were also assessed. Statistical analysis was performed using appropriate comparative tests, and a p-value ≤0.05 was considered statistically significant.

Results: Baseline characteristics were comparable between the two groups (p>0.05). At six months, a significantly higher proportion of patients in the statin group achieved LDL-C <70 mg/dL or ≥50% reduction compared with the non-statin group (77.3% vs. 56.0%, p=0.003). The mean reduction in LDL-C was also greater in the statin group (−38.2±21.4 mg/dL) than in the non-statin group (−26.1±23.8 mg/dL; p<0.001). A ≥50% reduction in LDL-C was observed in 48.0% of statin-treated patients compared with 29.3% in the non-statin group (p=0.012). Significant improvements were also observed in non-HDL cholesterol and triglyceride levels in the statin group (p<0.05). The decline in eGFR was smaller among statin users (p=0.045). Safety outcomes were similar in both groups.

Conclusion: Statin therapy significantly improved lipid parameters without increasing adverse events, supporting its role in cardiovascular risk management among CKD patients.

Keywords: Chronic kidney disease, Statins, LDL cholesterol, Dyslipidemia, Cardiovascular risk.

Introduction

Cardiovascular disease (CVD) accounts for 40–50% of deaths in patients with chronic kidney disease (CKD), which affects over 800 million people worldwide and is the ninth most common cause of death. [1] Dyslipidemia a hallmark of CKD manifests as hypertriglyceridemia, low HDL-cholesterol (HDL-C), and predominance of small dense LDL particles, exacerbated by uremic toxins, inflammation, and nephrotic proteinuria. [2] This atherogenic profile drives accelerated atherosclerosis, with CKD conferring 10-20-fold higher CVD risk compared to the general population, particularly in South Asian cohorts where metabolic syndrome prevalence exceeds 40%. [3]

Statins, the HMG-CoA reductase inhibitors, form the bedrock of CKD lipid therapy. A 2024 meta-analysis of 16 randomized controlled trials (RCTs) involving 3,594 non-dialysis CKD patients demonstrated significant reductions in total cholesterol, and triglycerides with statin therapy, with effects strengthening over time. [4] Cardiovascular benefits are well-established in early CKD: statins reduce major adverse cardiovascular events (MACE) by 25-41% in stages 3-5, as per SHARP and post-hoc analyses. [5] However, outcomes are neutral in dialysis populations, prompting nuanced KDIGO 2024 recommendations for statin initiation in eGFR 20-59 mL/min/1.73 m² but de-escalation consideration in ESRD. [6]

Despite maximal statins, 30-50% of CKD patients fail LDL-C targets (<70 mg/dL or ≥50% reduction), necessitating non-statins. [2] Ezetimibe augments LDL-C lowering by 20-25%; a 2025 quality improvement study reported 27% LDL-C and 24.8% non-HDL

reductions in CKD, mirroring non-CKD responses with comparable safety. [7] PCSK9 inhibitors (alirocumab, evolocumab) yield 50-60% LDL-C drops; FOURIER and ODYSSEY subgroups showed MACE reductions in eGFR <60 mL/min/1.73 m², with trends toward greater absolute benefits in advanced CKD. [8] Bempedoic acid is an ATP-citrate lyase inhibitor, with 18-25% reduction in LDL-C in statin-intolerant patients, and is safe in CKD by showing a CLEAR Outcomes with averaged LDL reduction of 23.2 mg/dL (MACE hazard ratio 0.85). [9]

Recent evidence has captured this: KDIGO 2024 supports the use of risk-specific non-HDL-C/non-LDL targets, with the use of ezetimibe/PCSK9/bempedoic add-on recommended when targets are not met. [6] The European lipid guideline (2024) also categories CKD as very high-risk with LDL-C goal of <55mg/dl. [10]

In Pakistan, the CKD has a prevalence of 18% and Ayub Teaching Hospital (ATH), Abbottabad which covers mountainous Khyber Pakhtunkhwa, has over 300 CKD/dialysis cases annually. [3] Local data have identified post-dialysis dyslipidemia in 60-70 percent of patients, but lipid therapy compliance and comparative effectiveness is unexplored. [11] The paper is a prospective study that evaluates the lipid normalization rate and safety of statins and non-statins (ezetimibe, PCSK9 neutralizers, bempedoic acid) in the CKD cohort of ATH, which fills the evidence gaps to maximize CVD prevention in this high-burden environment.

Methodology

The study is a prospective cohort study that will be carried out at the Department of Nephrology, Ayub Teaching Hospital (ATH), Abbottabad, Pakistan, during the period of January to December 2024. It was approved by the ATH Institutional Review Board (approval number [Ref.No.RC-EA-2023/109]) and adhered to an agreed-upon version of the Declaration of Helsinki (2013). Informed consent was signed by all the participants, and data confidentiality was ensured.

According to the Kidney Disease: Improving Global Outcomes (KDIGO) 2021 criteria, participants were individuals who were 18 years of age or older with chronic kidney disease (CKD) stages 2–5 (estimated glomerular filtration rate [eGFR] <60 mL/min/1.73 m² and/or signs of kidney damage persisting for ≥3 months). Eligible patients had been receiving monotherapy with either a statin or a non-statin lipid-lowering agent (ezetimibe, PCSK9 inhibitor, or bempedoic acid) for at least 3 months prior to baseline enrollment. Exclusion criteria encompassed acute kidney injury, combination lipid-lowering therapy, active liver disease, untreated hypothyroidism, nephrotic-range proteinuria (>3.5 g/day), pregnancy or lactation, and concurrent use of medications known to substantially affect lipid metabolism.

Using non-probability consecutive sampling, we enrolled patients from nephrology outpatient and inpatient services, targeting 150 participants (75 per group). The sample size was determined using OpenEpi version 3.01, assuming a minimum detectable mean difference of 20 mg/dL in low-density lipoprotein cholesterol (LDL-C) reduction (standard deviation 35 mg/dL, 80% power, α=0.05), based on data from prior CKD studies. Respondents were prospectively followed in 6 months (± 2 weeks), and regular visits were conducted (at baseline, 3 months, and 6 months) to assess treatment adherence and conduct laboratory tests.

Data were gathered through interviews with patients and medical record review at each visit using structured patient interviews. These were demographic variables (age, sex, body mass index, smoking status, CKD stage and duration), and clinical information (comorbidities (diabetes mellitus, hypertension, and ischemic heart disease); lipid-lowering therapy type, dose, and duration). The lipid profile (total cholesterol, triglycerides, LDL-C, high-density lipoprotein cholesterol), renal (serum creatinine, blood urea nitrogen, eGFR using CKD-EPI 2021 equation) and fasting blood glucose and liver (fasting blood) were measured by using standardized enzymatic tests on the fasting blood samples (≥12 hours). Safety parameters included creatine kinase, alanine aminotransferase, aspartate aminotransferase, and documentation of adverse events such as myalgia or rhabdomyolysis.

The statistical analysis was carried out with IBM SPSS version 25.0. For continuous variables, baseline characteristics were expressed as means ± standard deviations, and for categorical variables, as frequencies (percentages). The chi-square or Fisher's exact test was used for categorical data and the independent samples t-test or Mann-Whitney U test for continuous data in between-group comparisons. Wilcoxon signed-rank tests or paired t-tests were used to evaluate within-group differences from baseline. The primary outcome was the proportion of patients achieving LDL-C <70 mg/dL or ≥50% reduction from baseline at 6 months. Secondary outcomes included changes in LDL-C, non-high-density lipoprotein cholesterol, and triglycerides over time, as well as safety events. Between-group differences in changes were compared using analysis of covariance or Mann-Whitney U tests. Statistical significance was defined as a two-sided p-value less than 0.05.

Results

The study comprised 150 individuals with chronic renal disease, 75 in each of the statin and non-statin groups. Table I summarizes the participants' baseline clinical and demographic details. Patients on statin therapy had a mean age of 59.1±12.2 years, while the non-statin group had a mean age of 57.7±11.4 years (p=0.42). Male patients constituted 57.3% of the statin group and 52.0% of the non-statin group. The mean BMI was comparable between the groups (26.4±4.1 vs 26.1±3.9 kg/m²; p=0.69). Similarly, the prevalence of diabetes (64.0% vs 58.7%) and hypertension (82.7% vs 80.0%) did not differ significantly between the two groups. The distribution of CKD stages, baseline eGFR, and LDL-C levels were also comparable (p>0.05), indicating that the study groups were similar at baseline.

Table I: Distribution of Baseline Characteristics

Characteristics	Statin (n=75)	Non-Statin (n=75)	p-value
Age, years (mean ± SD)	59.1 ± 12.2	57.7 ± 11.4	0.42
Male sex, n (%)	43 (57.3)	39 (52.0)	0.52
BMI, kg/m² (mean ± SD)	26.4 ± 4.1	26.1 ± 3.9	0.69
Diabetes, n (%)	48 (64.0)	44 (58.7)	0.49
Hypertension, n (%)	62 (82.7)	60 (80.0)	0.69
CKD stage, n (%) 3 4 5	 32 (42.7) 25 (33.3) 18 (24.0)	 31 (41.3) 24 (32.0) 20 (26.7)	0.92
eGFR, mL/min/1.73 m² (mean ± SD)	32.1 ± 15.1	30.3 ± 14.1	0.46
LDL-C, mg/dL (mean ± SD)	128.5 ± 32.4	132.1 ± 34.7	0.51

The comparison of primary outcomes at six months is shown in Table II. A significantly higher proportion of patients in the statin group achieved LDL-C levels <70 mg/dL or a ≥50% reduction compared with those in the non-statin group (77.3% vs 56.0%; p=0.003). The mean reduction in LDL-C was significantly greater among statin users (−38.2±21.4 mg/dL) compared with the non-statin group (−26.1±23.8 mg/dL; p<0.001). In addition, nearly half of the patients receiving statins achieved a ≥50% reduction in LDL-C compared with 29.3% in the non-statin group (p=0.012).

Table II: Comparison of Primary Outcomes at 6 Months.

Primary Outcome	Statin (n=75)	Non-Statin (n=75)	p-value
LDL-C <70 mg/dL or ≥50% reduction, n (%)	58 (77.3)	42 (56.0)	0.003
LDL-C reduction, mg/dL (mean ± SD)	-38.2 ± 21.4	-26.1 ± 23.8	<0.001
≥50% LDL-C reduction, n (%)	36 (48.0)	22 (29.3)	0.012

Changes in secondary lipid parameters and renal function are summarized in Table III. Statin therapy resulted in significantly greater reductions in non-HDL cholesterol and triglyceride levels compared with non-statin therapy (p=0.008 and p=0.019, respectively). However, the increase in HDL-C was similar between the groups (p=0.81). A smaller decline in eGFR was observed in the statin group compared with the non-statin group (−1.8±4.2 vs −3.2±4.9 mL/min/1.73 m²; p=0.045).

Table III: Comparison of Secondary Lipid and Renal Changes at 6 Months

Parameters	Statin (n=75)	Non-Statin (n=75)	P-value
Non-HDL-C	-42.3 ± 24.1	-31.8 ± 26.5	0.008
Triglycerides	-48.2 ± 37.9	-35.4 ± 35.2	0.019
HDL-C	+2.4 ± 5.1	+2.1 ± 4.7	0.81
eGFR, mL/min/1.73 m²	-1.8 ± 4.2	-3.2 ± 4.9	0.045

Safety outcomes during the six-month follow-up are shown in Table IV. The overall frequency of adverse events was comparable between the statin and non-statin groups (18.7% vs 16.0%; p=0.68). Myalgia and elevated liver enzymes were infrequent and similar in both groups, and major adverse cardiovascular events were rare, indicating comparable safety profiles between the two treatment strategies.

Table IV: Comparison of Safety Outcomes at 6 Months

Safety Outcomes	Statin (n=75)	Non-Statin (n=75)	P-value
Any adverse event, n (%)	14 (18.7)	12 (16.0)	0.68
Myalgia, n (%)	5 (6.7)	3 (4.0)	0.72
ALT/AST >3× ULN, n (%)	2 (2.7)	2 (2.7)	1.00
MACE, n (%)	2 (2.7)	0 (0.0)	0.52†

ALT=alanine aminotransferase; AST=aspartate aminotransferase; ULN=upper limit of normal; MACE=major adverse cardiovascular event; †Kaplan-Meier log-rank test.

Discussion

The current study assessed the safety and effectiveness of statins in patients with chronic kidney disease (CKD) in comparison to non-statin lipid-lowering drugs. The results showed that the two groups' baseline clinical and demographic parameters were identical. The statin group's mean age was 59.1±12.2 years, while the non-statin group's was 57.7±11.4 years., with males constituting 57.3% and 52.0% of the groups, respectively. Additionally, diabetes and hypertension were highly prevalent in

both groups (64.0% vs 58.7% and 82.7% vs 80.0%, respectively). These findings are consistent with previous studies reporting that CKD frequently coexists with metabolic disorders such as diabetes and hypertension, which contribute to dyslipidemia and increased cardiovascular risk in these patients. [12–14] Studies from South Asia have similarly reported high rates of dyslipidemia among CKD patients, emphasizing the importance of lipid control in this population. [22,23]

The main finding of this study showed that patients on statin medication were far more likely than those on non-statin therapy to reach the LDL-C target (<70 mg/dL or ≥50% decrease) (77.3% vs. 56.0%, p=0.003). Also, the statistical difference between the mean changes in LDL-C between the statin group (−38.2±21.4 mg/dL) and non-statin group (−26.1+23.8 mg/dL) is statistically significant (p<0.001). This is in line with other large clinical trials that have compared statin therapy in CKD patients. Palmer et al. found that statin therapy in CKD patients led to a mean reduction of LDL-C level of about 35-40 mg/dl which is quite similar to the result in the statin group of the current study. [15] Similarly, the SHARP trial involving more than 9000 CKD patients demonstrated that statin-based therapy produced a reduction in LDL-C of approximately 33 mg/dL and significantly lowered the risk of major atherosclerotic events. [16]

The current research also showed a markedly higher decrease in non-HDL cholesterol and triglyceride levels in statin users relative to non-statin users. In particular, in the statin group, non-HDL cholesterol reduced by -42.3+24.1 mg/dl compared with -31.8+ 26.5 mg/dl in the non-statin group (p=0.008), and triglycerides by -48.2+37.9 mg/dl and -35.4+35.2 mg/dl, respectively (p=0.019). These results are in line with the prior reports that statins enhance various lipid fractions, by the inhibition of HMG-CoA reductase and decreased cholesterol production in the liver. It was also noted by Tonelli and Wanner that statins continue to be the mainstay treatment in the management of dyslipidemia in CKD because of their stable lipid-lowering effects and cardiovascular outcomes. [17] South Asian regional studies that have assessed the use of statin therapy in CKD patients have also reported similar improvements in lipid parameters.

The other interesting outcome of this research was a relatively lesser decrease in renal functionality in patients who underwent statin therapy than those who underwent non-statin therapy. The average eGFR decrease was -1.8+4.2 mL/min/1.73 m^2 in the statin group and -3.2 + 4.9 mL/min/1.73 m^2 in the non-statin group (p=0.045). Whereas the lipid lowering treatment is mainly prescribed to lower cardiovascular risk and not necessarily to slow down the progression of CKD directly, various studies have also proposed theoretical Reno protective benefits of statins. According to Hou et al., statin therapy can have a slight positive effect on renal function deterioration in CKD patients. [18] It has also been shown by observational studies that statins have led to better survival and lower progression to end-stage renal disease in CKD patients, but large randomized studies like the SHARP study show that the key effect of statins in CKD is on reduction of cardiovascular events and not on alteration of renal disease progression. [16,18]

Regarding safety outcomes, there was not a significant difference in the incidence of adverse events between the statin group and the non-statin group. Myalgia affected 6.7% and 4.0% of patients, respectively, whereas the overall incidence of adverse events was 18.7% in the statin group and 16.0% in the non-statin group. In both groups, elevated liver enzymes were uncommon, occurring in 2.7% of patients. These results are in line with earlier collaborative research that demonstrated statin medication is typically safe and well tolerated in patients with chronic kidney disease. [19,20] Regional studies have also reported low rates of statin-related adverse events among South Asian CKD populations.[21]

It is important to acknowledge certain limitations in contrast to these outcomes. The data' generalisability may be limited by the single-center methodology, and recall bias may have been introduced because medication adherence was determined by patient reporting. Larger multicenter longitudinal studies are recommended to further evaluate long-term cardiovascular and renal outcomes in CKD patients receiving lipid-lowering therapy.

Conclusion

In conclusion, statin therapy was associated with significantly greater improvement in lipid parameters among patients with chronic kidney disease compared with those not receiving statins. Patients in the statin group achieved higher rates of target LDL-C reduction and showed greater decreases in LDL-C, non-HDL-C, and triglyceride levels over the six-month follow-up period. While both groups showed a minor decline in renal function, patients taking statins experienced a relatively lower decline in eGFR. Importantly, there was not a noticeable rise in adverse events, and the safety profiles of the two groups were similar. According to these findings, statin therapy is a safe and effective way to improve lipid control in CKD patients. It can also help high-risk CKD patients reduce their cardiovascular risk.

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