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Effect of Statin Therapy on Lipoprotein (A) evels in Patients Post Percutaneous Coronary Intervention- Prospective Study

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Abstract:
Introduction: *Cardiovascular diseases are a leading cause of death worldwide, with coronary artery disease (CAD) accounting for a major proportion of deaths. Dyslipidaemia contributes significantly to CAD progression, and lipoprotein(a) [Lp(a)] has emerged as an independent risk factor for adverse cardiovascular outcomes. Percutaneous coronary intervention (PCI) is widely performed for CAD; however, residual risk persists due to lipid abnormalities. Statins are the cornerstone of lipid-lowering therapy post-PCI; their effect on Lp(a) remains uncertain. This study evaluated the impact of statin therapy on lipid parameters, including Lp(a) levels, in post-PCI patients.*
Aim: *To assess the effect of statin therapy on total cholesterol, LDL-C, HDL-C, triglycerides, and Lp(a) levels in patients following PCI.*
Materials and Methods: *This prospective study included 57 patients with dyslipidaemia aged 18–65 years who underwent PCI. The participants received atorvastatin (20 mg) or rosuvastatin (10 mg) for 90 days. Fasting lipid profiles and Lp(a) levels were measured at baseline and after therapy, with statistical significance set at $p<0.05$.*
Results: *Most patients were between 51 and 60 years (43.9%), and males constituted 56.1% of the study population. Coexisting diabetes and hypertension were present in 70.2% of the cases, reflecting a high-risk group. After 90 days of statin therapy, the mean total cholesterol significantly decreased from 190.16 ± 65.11 to 163.77 ± 60.91 mg/dL ($p<0.0001$), while LDL-C reduced from 131.37 ± 46.21 to 107.82 ± 46.30 mg/dL ($p<0.0001$). Triglyceride levels also showed a significant decrease from 145.00 mg/dL to 119.00 mg/dL ($p<0.0001$). In contrast, HDL-C levels remained largely unchanged, showing no significant variation. Importantly, Lp(a) levels rose significantly from 23.07 ± 5.56 to 31.86 ± 13.56 mg/dL ($p<0.0001$), indicating that although statins effectively control conventional lipids, they may worsen residual risk by elevating Lp(a) levels.*
Conclusion: *Statin therapy after PCI markedly improved total cholesterol, LDL-C, and triglyceride levels, confirming its central role in the secondary prevention of CAD. However, unchanged HDL-C levels and a significant increase in Lp(a) highlight an important therapeutic limitation.*
Keywords: *Statin therapy, Lipoprotein(a), Percutaneous coronary intervention, Dyslipidaemia, LDL-C, Residual cardiovascular risk*

1. INTRODUCTION

Cardiovascular diseases (CVD) remain the leading cause of mortality worldwide, with an estimated 17.8 million deaths per year. Nearly half of these deaths are due to coronary artery disease (CAD), which results from progressive atherosclerosis and its complications [1–4]. Lipid management is an essential part of both the prevention and treatment of CAD, as dyslipidaemia contributes to its development and progression. Among the different lipid fractions, lipoprotein(a) [Lp(a)], a genetically determined risk factor for atherosclerotic cardiovascular disease (ASCVD), has received the most attention [5,6].

Although Lp(a) and low-density lipoprotein (LDL) share structural similarities, Lp(a) also carries apolipoprotein(a), which exerts prothrombotic and proatherogenic effects. Elevated Lp(a) levels promote arterial plaque formation, impair fibrinolysis, and increase the risk of myocardial infarction, stroke, and peripheral artery disease. Despite the use of traditional lipid-lowering therapy, several studies have shown that elevated Lp(a) levels are associated with residual cardiovascular risk [5,7].

Patients with CAD are often treated with percutaneous coronary intervention (PCI) to restore blood flow. Although PCI relieves obstruction and improves myocardial perfusion, the risk of recurrent events

remains due to persistent lipid abnormalities. Elevated Lp(a) levels after PCI have been linked to higher rates of restenosis and recurrent cardiovascular events [8–10].

Following PCI, statins are commonly prescribed as first-line therapy for dyslipidaemia. By inhibiting HMG-CoA reductase, they reduce LDL cholesterol (LDL-C) and total cholesterol, thereby lowering the risk of future atherosclerotic events. Their anti-inflammatory and plaque-stabilising properties also provide cardiovascular benefits [9]. However, their effect on Lp(a) is limited, and in some cases, statins may slightly increase Lp(a) levels. This limitation is important in patients with elevated baseline Lp(a), as statins alone may not adequately reduce the related cardiovascular risk [10].

Guideline-based therapy recommends strict LDL-C targets for high-risk patients, and achieving these targets is associated with better outcomes. However, elevated Lp(a) remains difficult to manage. Although newer agents such as PCSK9 inhibitors and antisense oligonucleotides have shown effectiveness in lowering Lp(a), statins remain the most widely used and affordable option in clinical practice [7–10]. This study aimed to evaluate the effect of statin therapy on lipid parameters in PCI patients, including total cholesterol, LDL-C, HDL-C, triglycerides, and Lp(a). It further sought to determine whether statin therapy adequately addressed both conventional and residual lipid-related risks in post-PCI patients by assessing changes in lipoprotein levels and their relation to treatment goals.

2. MATERIALS AND METHODS

This three-month prospective study was conducted at SRM Medical College with 57 patients. Ethical approval was obtained from the Institutional Ethics Committee, and informed consent was secured from all participants before enrolment.

Sample size calculation

The sample size was calculated using the standard formula for proportion-based studies. With a prevalence (P) of 62.7% (≈63), an allowable error (d) of 12.6, and a 95% confidence level ($Z\alpha/2 = 1.96$), the required sample size was 57. The formula applied was $n = (Z\alpha/2^2 \times P \times Q) / d^2$, where $Q = 100 - P$.

Inclusion criteria

The study included patients aged 18–65 years with a confirmed diagnosis of dyslipidaemia based on lipid profile results, either admitted to the hospital or attending outpatient clinics.

Exclusion criteria

Patients aged < 18 years, those who did not provide informed consent, individuals with liver or kidney disease, familial hypercholesterolaemia, and those receiving non-statin therapy for dyslipidaemia were excluded from the study.

Methods

Basele information, including demographic details, medical history, comorbidities, current medications, and lifestyle factors, was collected from all participants. Fasting venous blood samples were collected at enrolment to measure total cholesterol, LDL-C, HDL-C, triglycerides, and Lp(a) using standard laboratory methods. After baseline assessment, the patients were started on statin therapy with either atorvastatin 20 mg or rosuvastatin 10 mg once daily. The treatment was continued for three months. At the end of the 3 months, fasting venous blood samples were collected again to repeat the lipid profile and Lp(a) estimations. All laboratory analyses were performed using uniform procedures to ensure reliability. Pre- and post-treatment values were compared to evaluate the effect of statin therapy on lipid and Lp(a) levels in post-PCI patients.

3. STATISTICAL ANALYSIS

The study utilised descriptive statistics, including mean, standard deviation, median, interquartile range, frequency, and percentage, to summarise the data. Differences in continuous variables between baseline and 90 days were assessed using paired t-tests. The Wilcoxon signed-rank test was applied to variables that did not follow a normal distribution. A p-value <0.05 was considered statistically significant using a two-tailed test. All analyses were performed using IBM SPSS Statistics v25.0.

4. RESULTS

Most of the patients were between 51 and 60 years of age (43.9%), followed by 24.6% who were below 50 years, 22.8% between 61 and 70 years, and above 71 years (8.8%). Regarding gender, 56.1% were

male and 43.9% were female. Regarding comorbidities, 70.2% of the patients had both diabetes mellitus (DM) and hypertension, while 29.8% had DM alone (Table 1).

Table 1: Distribution of study patients by age, gender, and comorbidities

Variable	Category	Frequency (%)
Age (years)	< 50	14 (24.6%)
	51–60	25 (43.9%)
	61–70	13 (22.8%)
	> 71	5 (8.8%)
Gender	Male	32 (56.1%)
	Female	25 (43.9%)
Comorbidities	DM only	17 (29.8%)
	DM + Hypertension	40 (70.2%)

The mean total cholesterol decreased from 190.16 ± 65.11 mg/dL at baseline to 163.77 ± 60.91 mg/dL, with a significant difference ($t=6.2$, $p<0.0001$). Similarly, LDL-C levels decreased from 131.37 ± 46.21 mg/dL to 107.82 ± 46.30 mg/dL ($t=7.287$, $p<0.0001$), indicating a marked improvement. Triglyceride levels also declined from a median of 145.00 mg/dL (range, 107–198) to 119.00 mg/dL (range, 83–167.5), showing a significant reduction ($z=-4.993$, $p<0.0001$). In contrast, HDL-C levels showed no significant change, with values of 42.11 ± 14.10 mg/dL at baseline and 41.00 ± 10.99 mg/dL after 90 days ($t=0.868$, $p=0.389$). Notably, Lp(a) levels increased significantly from 23.07 ± 5.56 mg/dL to 31.86 ± 13.56 mg/dL ($t=-5.203$, $p<0.0001$) (Table 2).

Table 2: Comparative changes in lipid parameters from baseline to 90 days of therapy

Parameter	(Mean ± SD)		t value/ z value	p value
	Baseline	90 Days		
Total Cholesterol (mg/dL)	190.16 ± 65.11	163.77 ± 60.91	t=6.2	<0.0001
LDL-C (mg/dL)	131.37 ± 46.21	107.82 ± 46.3	t=7.287	<0.0001
HDL-C (mg/dL)	42.11 ± 14.1	41.00 ± 10.99	t = 0.868	0.389
Triglycerides (mg/dL)	145.00 (107–198)	119.00 (83–167.5)	z = -4.993	<0.0001
Lp(a) (mg/dL)	23.07 ± 5.56	31.86 ± 13.56	t=-5.203	<0.0001

5. DISCUSSION

The present study was designed to evaluate the effect of statin therapy on lipid parameters, including total cholesterol, LDL-C, HDL-C, triglycerides, and Lp(a), in patients who have undergone PCI. Most patients were aged 51–60 years (43.9%), and the study population included 56.1% males and 43.9% females. Similarly, **Zhu et al.** reported a mean age of 65.9 ± 9.7 years, with 67.2% males [11]. **Suwa et al.** found that in patients with CAD after first PCI, the mean age was 64 years, with 78–86% male [9]. The demographic distribution highlights the predominance of middle-aged men, showing a higher CAD burden in this group.

In our study, 70.2% of patients had both diabetes and hypertension, whereas 29.8% had diabetes alone. Similarly, **Lin et al.** observed that females accounted for 17%, 24.7%, and 28.8% of the DM, HT, and DM+HT groups, respectively, whereas males accounted for 83.0%, 75.3%, and 71.2%, respectively [12]. **Lee et al.** reported that in patients who underwent PCI, diabetes was present in 31.4% of patients, while hypertension was reported in 87% of patients [13]. The high coexistence of diabetes and hypertension in patients undergoing PCI reflects their synergistic impact on atherosclerotic progression and cardiovascular risk.

Our study showed that the mean total cholesterol significantly decreased from 190.16 ± 65.11 mg/dL to 163.77 ± 60.91 mg/dL after 90 days ($p<0.0001$). Similarly, **Ballantyne et al., and Aslani et al.** confirmed

that statins reduced total cholesterol by –33.37 mg/dL in patients with cardiovascular disease [14,15]. Statin therapy consistently lowers total cholesterol levels, demonstrating strong lipid-lowering efficacy in both controlled trials and real-world clinical settings.

In our study, LDL-C decreased from 131.37 ± 46.21 mg/dL to 107.82 ± 46.30 mg/dL ($p<0.0001$). Similarly, **Ballantyne et al.** reported a reduction in LDL-C levels from 267 mg/dL to 157 mg/dL after 12 weeks, representing a 41.1% decline [14]. **Nasso et al.** showed that LDL-C levels fell from 155 ± 16 mg/dL to 114 ± 12 mg/dL with standard therapy [16]. **Burger et al.** reported a greater reduction with statin plus ezetimibe of LDL levels (−73 mg/dL, −52.1%) than with statin alone (−43 mg/dL, −42.2%; $p<0.001$) [17]. **Harris DE et al.** found that only 5.3% of PCI patients achieved LDL-C <1.0 mmol/L [18]. Statins provide substantial LDL-C reduction, although many patients fail to reach optimal targets, supporting combination therapy for high-risk groups.

Our study showed that triglyceride levels declined significantly from 145.00 mg/dL to 119.00 mg/dL ($p<0.0001$). Similarly, **Park et al.** reported a reduction of triglycerides from 269.8 mg/dL to 145.5 mg/dL after 8 weeks of fenofibrate plus statin ($p<0.0001$) [19]. **Maki et al.** found that pitavastatin led to a 23.3% decrease in patients with elevated triglycerides [20]. Triglyceride reduction with statins shows their role in improving residual lipid abnormalities.

In our study, HDL-C remained unchanged, from 42.11 ± 14.10 mg/dL to 41.00 ± 10.99 mg/dL ($p=0.389$). Similarly, **Yang et al.** found no significant changes in HDL-C patients with ACS post-PCI [21]. **Yun et al.** reported that rosuvastatin increased HDL-C by 1.1 ± 9.8 mg/dL, with increases in 56.1% and decreases in 43.9% of patients [22]. Statins have minimal or variable effects on HDL-C, suggesting that their cardiovascular benefits are largely mediated through LDL-C lowering.

Our study showed that Lp(a) levels increased from 23.07 ± 5.56 mg/dL to 31.86 ± 13.56 mg/dL after 90 days ($p<0.0001$). Similarly, **Tsimikas et al.** reported Lp(a) increases with statins, with pravastatin rising 11.6–20.4% and atorvastatin 18.7–24.2% [23]. **Randall et al.** showed similar increases in Lp(a) in obese African Americans after lifestyle modifications [24]. **Ndrepepa et al.** found LDL-C was not independently associated with 1-year mortality (HR 1.03, 95% CI 0.90–1.17; $p=0.294$) [25]. Statins increase Lp(a) levels, a risk factor for adverse cardiovascular events, highlighting the need for targeted therapies beyond LDL-C lowering.

Our study showed that statin therapy after PCI significantly lowered total cholesterol, LDL-C, and triglyceride levels, whereas HDL-C levels remained unchanged, and Lp(a) levels increased. The findings show the powerful LDL-C-lowering effect of statins but also emphasise the limitations regarding residual risk factors. Overall, although statins remain the cornerstone of therapy, additional strategies may be required for comprehensive lipid management in high-risk patients.

Limitation(s)

The study had a relatively small sample size and short follow-up duration, which may limit the generalisability of the results. The study was conducted at a single centre, which could have introduced selection bias. Factors such as dietary habits, genetic influences, and adherence to therapy were not fully controlled, which may have affected lipid changes.

6. CONCLUSION(S)

Our study shows that after PCI, statin therapy effectively reduced triglycerides, LDL-C, and total cholesterol, confirming its important role in secondary prevention of CAD. However, Lp(a) levels increased and HDL-C remained unchanged, reflecting a key limitation in addressing all lipid-related risks. These findings support the strong LDL-C–lowering effect of statins but also their tendency to raise Lp(a) and their minimal impact on HDL-C. While statins are essential for cholesterol control, they may not fully eliminate residual cardiovascular risk. Additional therapies targeting Lp(a) and other unmet lipid parameters are needed to improve long-term outcomes in post-PCI patients.

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