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Original Research Article

Impact of Statin Treatment On Liver Enzyme Levels in Patients with Dyslipidemia

Joshi Abhishek[1], Modi Vansh Kanaiyalal[2], Modi Shraddhaben Kanaiyalal[3]

[1]MBBS, Odessa National Medical University, Ukraine
[2]MBBS, Odessa National Medical University, Ukraine
[3]Tutor, Department of Microbiology, GMERS Medical College, Patan, Gujarat, India

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Corresponding Author: Modi Shraddhaben Kanaiyalal
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Abstract:

Background: Dyslipidemia frequently coexists with non-alcoholic fatty liver disease (NAFLD) and cardiovascular disease, contributing to increased morbidity and mortality. While statins are widely prescribed to manage lipid abnormalities and reduce cardiovascular risk, their effect on liver enzyme levels in dyslipidemic patients with NAFLD remains incompletely understood. Evaluating this effect is critical to ensure both efficacy and safety of statin therapy in this high-risk population.

Methods: This prospective observational study enrolled 146 adult patients aged 18–80 years with dyslipidemia, NAFLD, and cardiovascular disease at a tertiary care hospital over one year. Patients receiving statins (n = 88) were compared with non-statin users (n = 58). Liver enzymes (ALT, AST) and lipid profiles (total cholesterol, LDL-C) were measured at baseline and follow-up. Demographic, clinical, and treatment-related data were also collected.

Results: At baseline, ALT and AST levels were similar between the statin and non-statin groups (43.1 ± 18.0 vs 40.1 ± 15.9 U/L and 38.0 ± 14.9 vs 35.2 ± 13.1 U/L, respectively; $p > 0.1$). After follow-up, statin therapy significantly reduced ALT (36.0 ± 14.0 U/L; $p < 0.01$) and AST (31.1 ± 11.7 U/L; $p = 0.04$), whereas non-statin patients showed minimal change. Total cholesterol decreased from 210.5 ± 30.9 to 180.9 ± 24.6 mg/dL ($p < 0.01$) and LDL-C from 135.4 ± 28.7 to 104.6 ± 21.0 mg/dL ($p < 0.01$) in the statin group, with no significant reductions in the non-statin group. Statins were well tolerated, with only minor side effects reported.

Conclusion: Statin therapy significantly improves liver enzyme levels and lipid profiles in dyslipidemic patients with NAFLD and cardiovascular disease. These results support the dual hepatic and cardiovascular benefits of statins in this population.

Keywords: Dyslipidemia, NAFLD, Statins, Liver Enzymes, Cardiovascular Disease.

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Introduction

Non-alcoholic fatty liver disease (NAFLD) is a common metabolic liver disorder characterized by excessive fat accumulation in the liver, independent of significant alcohol consumption. It is frequently observed in individuals with metabolic abnormalities such as obesity, type 2 diabetes mellitus, and dyslipidemia [1]. NAFLD represents a spectrum of hepatic involvement, ranging from simple steatosis to more advanced inflammatory forms such as non-alcoholic steatohepatitis (NASH), which may progress to cirrhosis and liver failure if left unmanaged [2]. The growing global prevalence of metabolic syndrome has led to NAFLD being recognized as one of the most prevalent chronic liver diseases worldwide, emphasizing the need for early identification and appropriate management strategies [1,3].

The terminology of NAFLD has recently evolved to metabolic dysfunction–associated steatotic liver disease (MASLD), highlighting its strong association with metabolic risk factors including insulin resistance, obesity, dyslipidemia, and diabetes mellitus [4]. MASLD is now considered both a hepatic and systemic condition, with cardiovascular disease (CVD) being the leading cause of morbidity and mortality among affected individuals. Shared pathogenic mechanisms such as chronic inflammation, oxidative stress, and atherogenic dyslipidemia contribute to the close interrelationship between MASLD and cardiovascular disease [5].

NAFLD commonly coexists with dyslipidemia and cardiovascular disease, creating a complex clinical scenario in which each condition may negatively

influence the other. Obesity, insulin resistance, and abnormal lipid metabolism are common risk factors for both NAFLD and CVD, and the presence of hepatic steatosis has been associated with an increased risk of atherosclerosis and adverse cardiovascular outcomes [6]. Conversely, alterations in lipid handling and systemic inflammation associated with cardiovascular disease may further aggravate hepatic injury [7].

Statins are widely prescribed lipid-lowering agents and form the cornerstone of dyslipidemia management. By inhibiting 3-hydroxy-3-methylglutaryl–coenzyme A (HMG-CoA) reductase, statins effectively reduce low-density lipoprotein cholesterol (LDL-C) levels and significantly lower the risk of cardiovascular events such as myocardial infarction and stroke. In addition to their lipid-lowering properties, statins exert pleiotropic effects including improvement in endothelial function and reduction of systemic inflammation [8].

Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are routinely used biomarkers of hepatocellular injury and are often elevated in patients with NAFLD. Elevated liver enzyme levels reflect underlying hepatic inflammation and are frequently encountered in dyslipidemic individuals with metabolic liver disease [9]. However, the impact of statin therapy on liver enzymes remains a subject of clinical concern and debate. While several studies have reported reductions in ALT and AST levels following statin treatment, others have described mild and transient elevations, raising questions regarding hepatic safety in routine clinical practice [10].

Despite the widespread use of statins in dyslipidemic patients, evidence regarding their effects on liver enzymes, particularly in those with underlying NAFLD or MASLD, remains inconsistent. Furthermore, data from South Asian populations are limited. Therefore, the present study aims to evaluate the effect of statin therapy on liver enzymes in dyslipidemic patients, providing clinically relevant insights into the hepatic safety profile of statins in this high-risk population.

Materials and Methods

Study Design, Setting, and Duration: This prospective observational study was conducted at a tertiary care hospital over a period of one year. The study aimed to evaluate the effect of statin therapy on liver enzymes in patients with dyslipidemia and coexisting cardiovascular disease and non-alcoholic fatty liver disease (NAFLD).

Study Population and Sample Size: A total of 146 patients were enrolled using a non-probability consecutive sampling method. Adult patients aged 18–80 years with a confirmed diagnosis of cardiovascular disease, including ischemic heart disease, hypertension, hyperlipidemia, or heart failure, were considered eligible. All participants had a documented diagnosis of NAFLD based on imaging modalities such as ultrasonography, computed tomography, or magnetic resonance imaging, or liver biopsy where available. Inclusion required ongoing statin therapy as part of routine clinical management for dyslipidemia or cardiovascular disease.

Exclusion Criteria

Patients with advanced liver disease, including cirrhosis or hepatocellular carcinoma, were excluded from the study. Individuals with significant alcohol consumption, viral hepatitis (hepatitis B or C), severe renal impairment (estimated glomerular filtration rate <30 mL/min/1.73 m²), or known contraindications to statin therapy, such as severe myopathy or significant hepatic or renal dysfunction, were also excluded. Pregnant or breastfeeding women and patients with serious comorbid conditions that could confound study outcomes were not included.

Data Collection and Study Variables

Baseline and follow-up data were collected through clinical evaluation, laboratory investigations, and imaging records. Demographic variables included age, sex, body mass index, comorbidities, and smoking status. Laboratory assessments focused on liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase. Details of statin therapy, including the type of statin, dosage, frequency, and duration of use, were documented along with concurrent cardiovascular medications. Cardiovascular parameters such as blood pressure and lipid profile—total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides—were also recorded. The primary outcome measure was the change in liver enzyme levels (ALT and AST) following statin therapy.

Statistical Analysis: Data analysis was performed using SPSS version 20. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. A p-value of less than 0.05 was considered statistically significant.

Results

The baseline demographic and clinical characteristics were comparable between the statin and non-statin groups, with no statistically significant differences observed across key variables. Mean age, sex distribution, and duration of disease were similar in both groups, indicating adequate baseline matching. The prevalence of

ischemic heart disease, heart failure, diabetes mellitus, hypertension, and obesity did not differ significantly between the groups, suggesting that the two cohorts were clinically comparable at study entry (Table 1).

Table 1: Baseline Demographic and Clinical Profile of the Study Population

Characteristic	Statin Group (n = 88)	Non-Statin Group (n = 58)	p-value
Age (years, mean ± SD)	56.9 ± 10.1	55.2 ± 10.4	0.15
Sex (Male/Female, n, %)	52 (59%) / 36 (41%)	32 (55%) / 26 (45%)	0.89
Duration of Disease (years, mean ± SD)	8.0 ± 4.0	7.7 ± 4.3	0.13
Ischemic Heart Disease, n (%)	61 (69%)	38 (66%)	0.93
Heart Failure, n (%)	17 (19%)	11 (19%)	0.74
Diabetes Mellitus, n (%)	44 (50%)	27 (47%)	0.95
Hypertension, n (%)	62 (70%)	41 (71%)	0.81
Obesity, n (%)	46 (52%)	30 (52%)	0.69

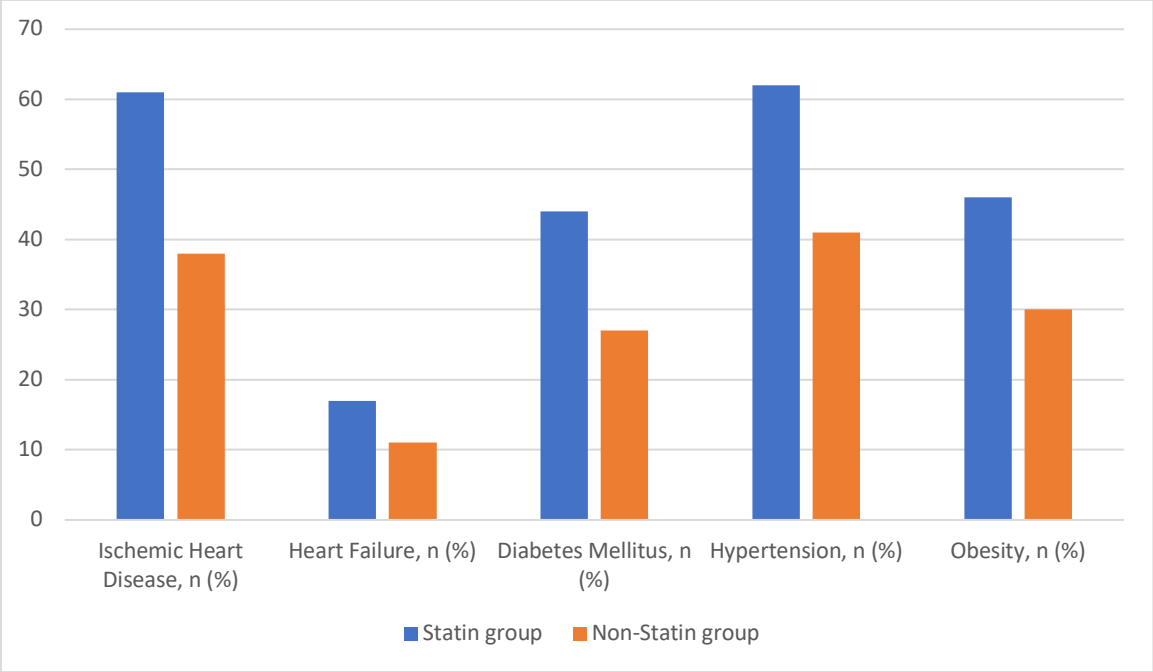

Figure 1: Patient distribution as per clinical profile.

At baseline, serum ALT and AST levels were comparable between the statin and non-statin groups, with no statistically significant differences observed. At follow-up, however, patients receiving statin therapy demonstrated a significant reduction in both ALT and AST levels compared with the non-statin group. These findings indicate an overall improvement in liver enzyme profiles among statin users, while enzyme levels remained relatively unchanged or higher in patients not receiving statins (Table 2).

Table 2: Comparison of Liver Enzyme Levels at Baseline and Follow-Up Between Study Groups

Parameter	Statin Group (n = 88)	Non-Statin Group (n = 58)	p-value	t-value
ALT (U/L) – Baseline	43.1 ± 18.0	40.1 ± 15.9	0.21	1.26
ALT (U/L) – Follow-Up	36.0 ± 14.0	40.0 ± 15.6	<0.01	−2.54
AST (U/L) – Baseline	38.0 ± 14.9	35.2 ± 13.1	0.18	1.35
AST (U/L) – Follow-Up	31.1 ± 11.7	35.8 ± 13.9	0.04	−2.09

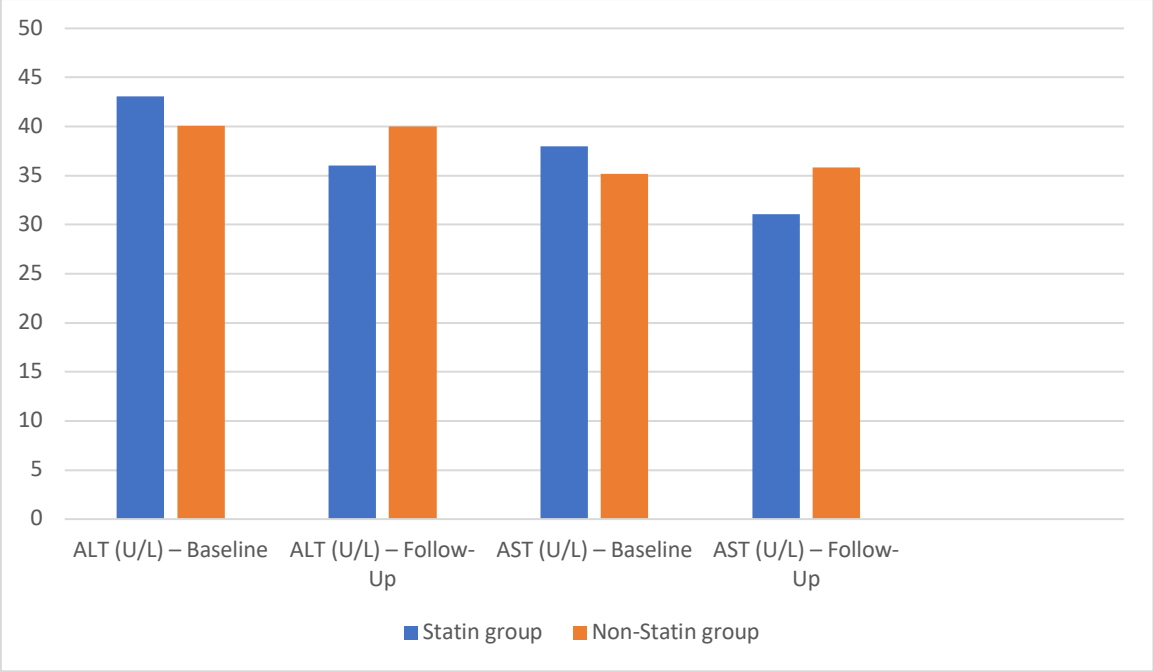

Figure 2: Comparison of Liver Enzyme Levels at Baseline and Follow-Up Between Study Groups.

Baseline lipid parameters revealed significantly higher total cholesterol and LDL-C levels in the statin group compared with the non-statin group. Following follow-up, patients in the statin group showed a marked reduction in total cholesterol and LDL-C levels, whereas changes in the non-statin group were less pronounced and not statistically significant. These results highlight the effectiveness of statin therapy in improving lipid profiles among dyslipidemic patients (Table 3).

Table 3: Changes in Lipid Parameters at Baseline and Follow-Up in Statin and Non-Statin Groups

Lipid Parameter (mg/dL)	Statin Group (n = 88)	Non-Statin Group (n = 58)	p-value	t-value
Total Cholesterol – Baseline	210.5 ± 30.9	196.7 ± 27.8	<0.01	3.98
Total Cholesterol – Follow-Up	180.9 ± 24.6	193.8 ± 26.5	0.23	−1.21
LDL-C – Baseline	135.4 ± 28.7	121.1 ± 26.5	<0.01	4.47
LDL-C – Follow-Up	104.6 ± 21.0	118.6 ± 24.5	0.18	−1.36

Abbreviation: LDL-C, low-density lipoprotein cholesterol

Discussion

This prospective observational study evaluated the impact of statin therapy on liver enzyme levels in dyslipidemic patients with coexisting cardiovascular disease (CVD) and non-alcoholic fatty liver disease (NAFLD). The findings demonstrate that statin use was associated with a significant improvement in hepatic biochemical markers, particularly alanine aminotransferase (ALT) and aspartate aminotransferase (AST), supporting the potential hepatic benefits of statins beyond their established role in cardiovascular risk reduction. These results suggest that statins may exert dual benefits by effectively managing dyslipidemia while also contributing to improved liver enzyme profiles in this high-risk population [11].

At baseline, elevated ALT and AST levels were observed in both statin and non-statin groups, which is consistent with the metabolic and inflammatory milieu commonly seen in patients with NAFLD. Over the follow-up period, patients receiving statin therapy demonstrated a meaningful reduction in liver enzyme levels, whereas no comparable improvement was noted among those not receiving statins. Specifically, ALT and AST levels declined significantly in the statin group over 12 months, reflecting an overall reduction in hepatocellular injury. These improvements may be attributed to the pleiotropic effects of statins, including their anti-inflammatory, antioxidant, and lipid-modulating properties, which extend beyond cholesterol lowering alone [12].

Concerns regarding statin-associated hepatotoxicity have historically limited their use in patients with underlying liver disease. However, the findings of this study indicate that statins were well tolerated and did not result in clinically significant liver injury in patients with mild to moderate NAFLD. The absence of severe elevations in liver enzymes or treatment-limiting hepatotoxicity aligns with growing evidence that statins can be safely prescribed in patients with metabolic liver disease [13]. This reinforces current clinical guidance that

routine discontinuation of statins due to mild liver enzyme elevations may be unnecessary.

In addition to favorable effects on liver enzymes, statin therapy resulted in significant improvements in lipid parameters among dyslipidemic patients. Notable reductions in total cholesterol and low-density lipoprotein cholesterol (LDL-C) were observed in the statin group during follow-up, confirming the efficacy of statins in achieving lipid control in patients with concurrent CVD and NAFLD. These improvements are clinically relevant, as elevated LDL-C and total cholesterol are key contributors to atherosclerosis and adverse cardiovascular outcomes [14]. Effective lipid lowering may also indirectly benefit hepatic metabolism by reducing lipid influx and lipotoxicity within the liver.

The observed improvement in liver enzymes among statin users may be explained by reductions in hepatic inflammation, oxidative stress, and lipid accumulation, leading to improved hepatocyte integrity and reduced enzyme leakage into the circulation. Statins have also been shown to improve endothelial function and modulate inflammatory pathways, which may further contribute to improved hepatic and systemic metabolic profiles [15,16]. Importantly, statin therapy was associated with minimal adverse effects in the present cohort, with only mild and transient symptoms such as muscle discomfort or gastrointestinal intolerance reported in a small proportion of patients [17,18].

Overall, the findings of this study support the safe and effective use of statins in dyslipidemic patients with NAFLD and cardiovascular disease. By improving both lipid parameters and liver enzyme levels, statins may offer a comprehensive therapeutic approach in this metabolically vulnerable population. These results add to the growing body of evidence supporting the hepatic safety of statins and highlight their potential role in the integrated management of dyslipidemia and NAFLD [19].

Conclusion

This study demonstrates that statin therapy is both effective and safe in dyslipidemic patients with coexisting cardiovascular disease and non-alcoholic fatty liver disease. Statin use was associated with significant reductions in serum liver enzymes (ALT and AST) over the follow-up period, indicating an improvement in hepatic biochemical status without evidence of clinically significant hepatotoxicity. In addition, statins effectively improved lipid parameters, reinforcing their central role in cardiovascular risk reduction. These findings support the continued and judicious use of statins in dyslipidemic patients with NAFLD and highlight their potential dual benefit in addressing both metabolic liver dysfunction and cardiovascular disease. Further large-scale and long-term studies are warranted to confirm these findings and explore their impact on long-term hepatic outcomes.

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