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A PROSPECTIVE OBSERVATIONAL STUDY OF ROLE AND MANAGEMENT OF STATINS IN CORONARY ARTERY DISEASE

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Abstract

Introduction: statins therapies are a cornerstone in the management of coronary artery disease (CAD), largely due to the prominent role of lipids and triglycerides play in the pathophysiology of the disease. HMG-CoA enzyme inhibitors are integral to the management of CAD ***Aim:*** the study aims to evaluate the real-world efficacy, adverse effect, symptoms, co-morbidities, and lipid profile of HMG-CoA enzyme inhibitors in CAD management. ***Methods:*** a meticulously designed prospective observational study was conducted on patients diagnosed with CAD, including acute coronary syndrome (ACS), Anterior Wall Myocardial Infarction (AWMI), unstable angina, myocardial infraction (MI). participants were stratified based on the prescribed HMG-CoA enzyme inhibitors. Key clinical endpoints, such as side effects, adverse effect, symptoms, co-morbidities, lipid profile and all-cause mortality were assessed over a structured 1-month follow-up period. ***Result:*** A total of 100 patients were enrolled. After 1 month of taking HMG-CoA enzyme inhibitors side effects, with muscle pain, weakness, headache, stomach pain the majority of recurring symptoms were observed in patients aged 41-80 years. The risk-benefit equilibrium of these agents underscores the necessity of precision medicine in tailoring statins therapy for CAD patients. ***Conclusion:*** HMG-CoA enzyme inhibitors integral to contemporary CAD management, with Atorvastatin and Rosuvastatin being most commonly used drugs in this disease. In this study, the adverse effects of statins are mild and transient. Fewer patients experienced sleeplessness after using these drugs, and overall, symptoms and side effects were relatively manageable. However, a significant portion of patients experienced side effects after taking HMG-CoA enzyme inhibitors in the management of coronary artery disease

Keywords: Prophylaxis**,** CORONARY ARTERY DISEASE**,** Hyperlipidaemia**,** HMG-CoA reductase, Statins, Deposition of cholesterol and lipids, hypolipidemic drugs, lipid profile in CAD treatment.

INTRODUCTION

CAD is also known as atherosclerotic heart disease, coronary atherosclerosis, coronary arteriosclerosis, coronary heart disease, Hyperlipidaemia, Deposition of cholesterol and lipids, Statins. Coronary artery disease (CAD) is a common type of heart disease. It affects the main blood vessels that supply blood to the heart, called the coronary arteries.

Coronary artery disease is caused by the buildup of fats, cholesterol and other substances in and on the walls of the heart arteries. This condition is called atherosclerosis. The buildup is called plaque. Plaque can cause the arteries to narrow,

blocking blood flow. The plaque also can burst, causing a blood clot. Abnormal accumulation of lipids or fatty substances and fibrous tissues in the vessels or walls of vessels. These substances block's or narrows the blood vessels there by reducing the blood flow to the myocardium and resulting in coronary artery disease. The formation of plaque is generally called as atherosclerosis. The main cause for myocardial infraction is coronary artery disease. A narrowing of the coronary arteries that prevents adequate blood supply to the heart muscle is called coronary artery disease. Usually caused by atherosclerosis, it may progress to the point where the heart muscle is damaged due to lack of blood supply. Such damage may result in infarction, arrhythmias, and heart failure.

HMG-CoA reductase inhibitors (statins) are lipid-lowering medications used in the primary and secondary prevention of coronary heart disease. This activity reviews the indications, contraindications, and mechanism of action of statins for the management of coronary heart disease and familial dyslipidaemias. This activity will cover the indications, contraindications, activity, adverse events, and other critical elements of statin therapy, and highlight the crucial role of the interprofessional team in the management of patients with clinically significant atherosclerotic cardiovascular disease or individuals with risk factors for heart disease who can benefit from statin therapy.

Hydroxymethylglutaryl-CoA (HMG-CoA) reductase inhibitors, also known as "statins," are used adjunctively to diet and exercise to treat hypercholesterolemia by lowering total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG) concentrations while increasing high-density lipoprotein cholesterol (HDL-C) concentrations. The approved FDA indications vary slightly between each statin but generally are indicated for the treatment and/or prevention of primary and secondary prevention clinical atherosclerotic cardiovascular disease (ASCVD) (e.g., myocardial infarction or stroke). Conversion of 3-hydroxy-3-methyl glutaryl-CoA (HMG-CoA) to mevalonate by HMG-CoA reductase in the hepatocytes is the first and rate-limiting step in cholesterol biosynthesis.

Statins competitively inhibit HMG-CoA reductase enzyme. Statins bind to the active site of the enzyme and induce a conformational change in its structure, thus reducing its activity. Also, the binding affinity of statins for HMG-CoA reductase is 10,000 times higher than the substrate (HMG-CoA), thus preventing the action of the enzyme and reducing the intracellular synthesis of cholesterol. Statins have a significant impact on lowering cholesterol since most of the circulating plasma cholesterol comes from the internal synthesis in hepatocytes rather than the diet. The reduced intracellular concentrations of cholesterol in hepatocytes secondary to statin use activate the proteases that cleave membrane-bound sterol regulatory element-binding proteins (SREBP), which further migrate to the nucleus and bind to sterol response elements. This binding results in increased transcription of the LDL receptor, which translocate to the liver cell membrane. The LDL and VLDL particles in plasma bind to the LDL receptors and endocytose in hepatocytes, where their cholesterol component gets processed into bile salts, which are then excreted or recycled. This process increases the catabolism of LDL and VLDL cholesterol and results in further reduction of plasma cholesterol concentrations.

Statins reduce the plasma concentrations of total cholesterol, LDL-C, VLDL-C, triglycerides, apo-B, and increase the plasma concentrations of HDL-C. Apart from lowering lipid concentrations, statins also have cardiovascular protective effects (pleiotropic effects), which are primarily because of the inhibition of the production of prenylated proteins (mainly farnesyl pyrophosphate and geranylgeranyl pyrophosphate) in the cholesterol biosynthetic pathway. Statins prevent cardiovascular disease progression via the following mechanisms.Plaque stabilization: Coronary artery plaque rupture predisposes to acute coronary syndrome. Statins maintain the integrity of the fibrous cap of atherosclerotic plaque, inhibit the proliferation of macrophages, and decrease the expression of matrix metalloproteinases (MMP).Reduces inflammation: Inflammation plays an essential role in atherosclerotic plaque rupture. Statins reduce the level of pro-inflammatory cytokines (TNF-a, IL-6, IL-8) and decrease the concentration of CRP.Improve endothelial function: Statins increase eNOS activity within the endothelial cells resulting in vasodilation and thus improving myocardial blood flow.Decreased thrombogenicity: Statins decrease the activity of platelets and reduce thromboxane A2 synthesis.

METHODOLOGY

The study was conducted at Apollo Hospital, Kakinada, over a six-month period, from August 2024 to February 2025, with a sample size of 100 cases. It was a prospective observational study involving both male and female patients, including geriatrics, aged 20-80 years, who are receiving hypolipidemic drugs (statins) data were collected on patient demographics, lab parameters (LDL, HDL, total cholesterol levels were monitored during a 1 month follow up. The Descriptive statistics were used for data analysis and findings were represented through bar graphs.

RESULTS

Among patients taking HMG-CoA enzyme inhibitors, 39% are on Atorvastatin, 37% are on Rosuvastatin and 24% are on Atorvastatin and Rosuvastatin**.** After taking HMG-CoA enzyme inhibitors, patients (73% male and 27% female) experienced adverse effects, among these patients, most common adverse effects are 28% experienced headache, 21%

experienced muscle pain, 20% experienced weakness. Furthermore 80% of patients have normal range of low-density lipoprotein cholesterol and 20% of patients having high LDL.

Table 1:Frequency table of statins groups

STATINS	No. of patients	Frequency
Atorvastatin	39	39%
Rosuvastatin	37	37%
Atorvastatin + Rosuvastatin	24	24%

STATINS WISE DISTRIBUTION:

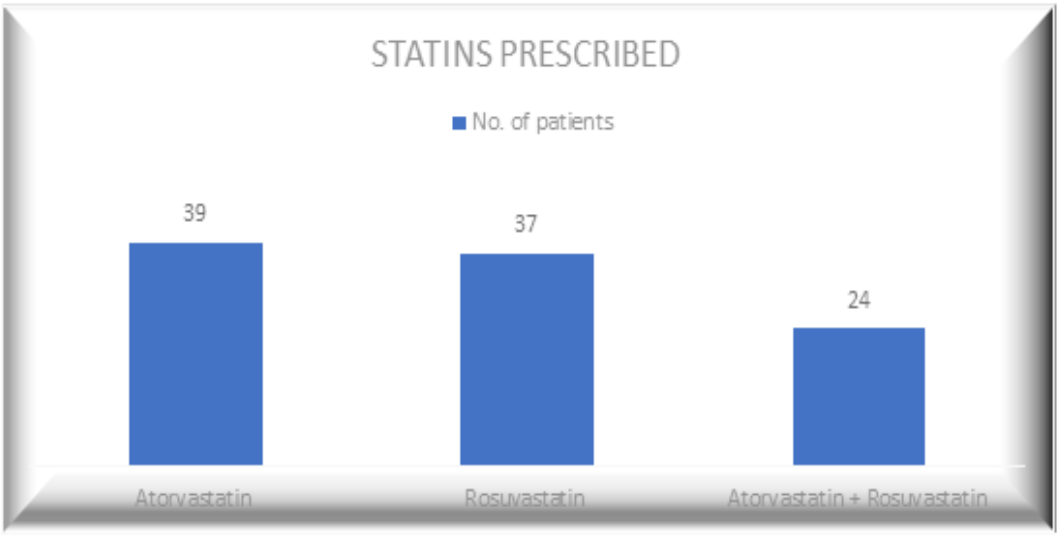


Table 2: Frequency table of type of CAD groups

Type of CAD	No. of patients	Frequency
AWMI	13	13%
ACS	22	22%
ACS with AWMI	11	11%
ACS with IWMI	03	03%
ACS with NSTEMI	01	01%
ACS with unstable angina	07	07%
Acute MI	02	02%
CAD	24	24%
CAD with AWMI	02	02%
CAD with IWMI	03	03%
IWMI	01	01%
Unstable angina	10	10%

Type of CAD WISE DISTRIBUTION

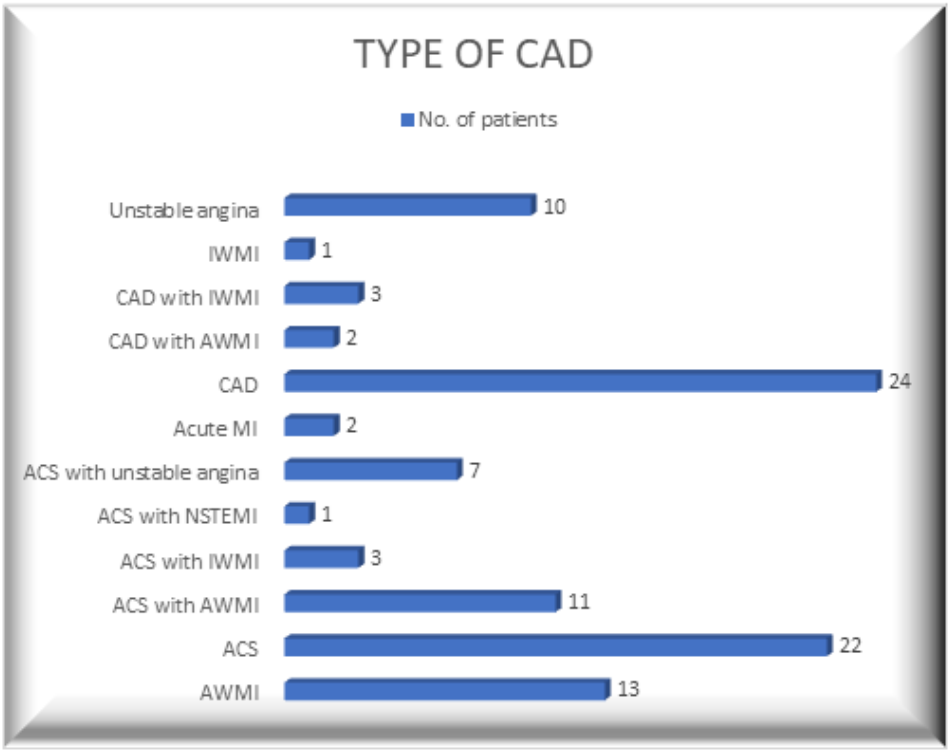


Table 3: Frequency table of adverse effects groups

TYPE	No of patients	Frequency
Muscle pain	21	21%
Drowsiness	15	15%
Heart burn	17	17%
Headache	28	28%
Weakness	20	20%
Stomach upset	12	12%
Constipation	05	05%
Joint pain	02	02%
Difficulty in falling sleep	10	10%
Nausea	11	11%
Slow heartrate	01	01%
Ankle swelling	02	02%

ADVERSE EFFECTS WISE DISTRIBUTION:

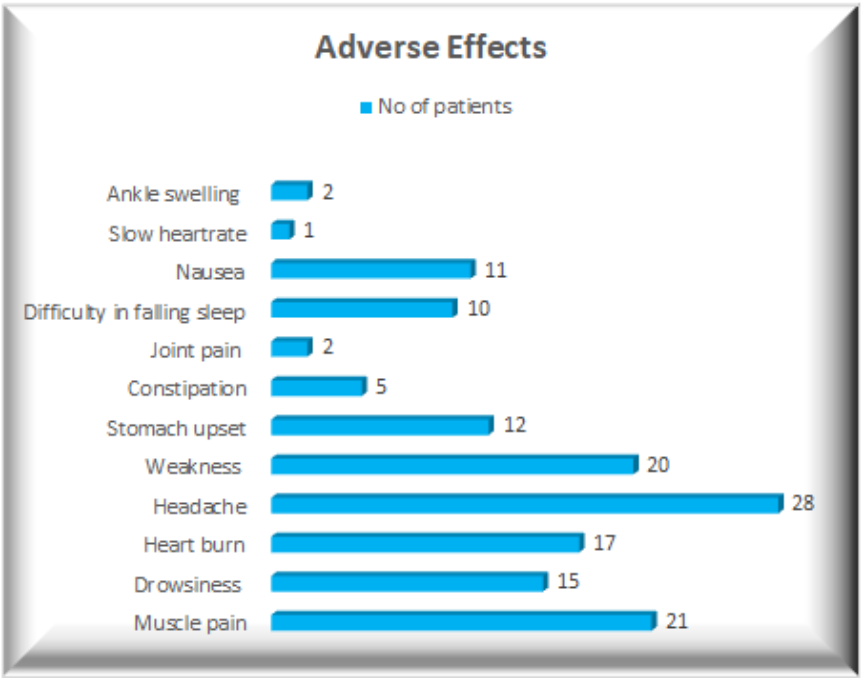
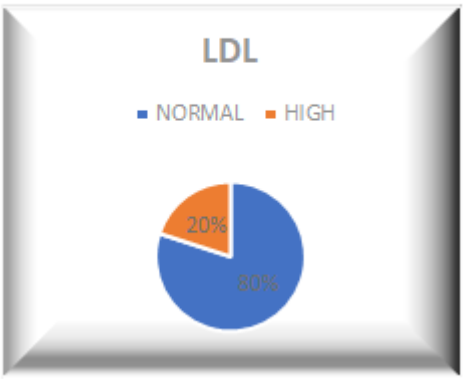


Table 4: Frequency table of lipid profile groups

Lipid profile		No of patients	Frequency
LDL	Normal	80	80%
	High	20s	20%
HDL	Low	13	13%
	Normal	87	87%

LIPID PROFILE WISE DISTRIBUTION:



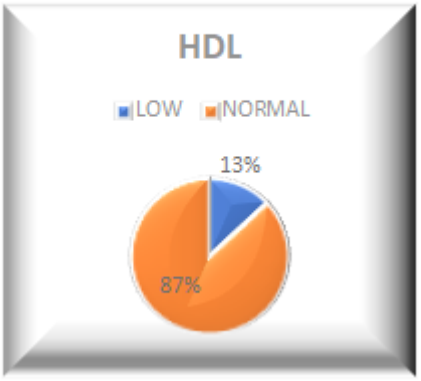


Table 5: Frequency table of total cholesterol groups

TAL CHOLESTEROL	. OF PATIENTS	EQUENCY
rmal		%
rderline high		%
gh		%

TOTAL CHOLESTEROL WISE DISTRIBUTION:

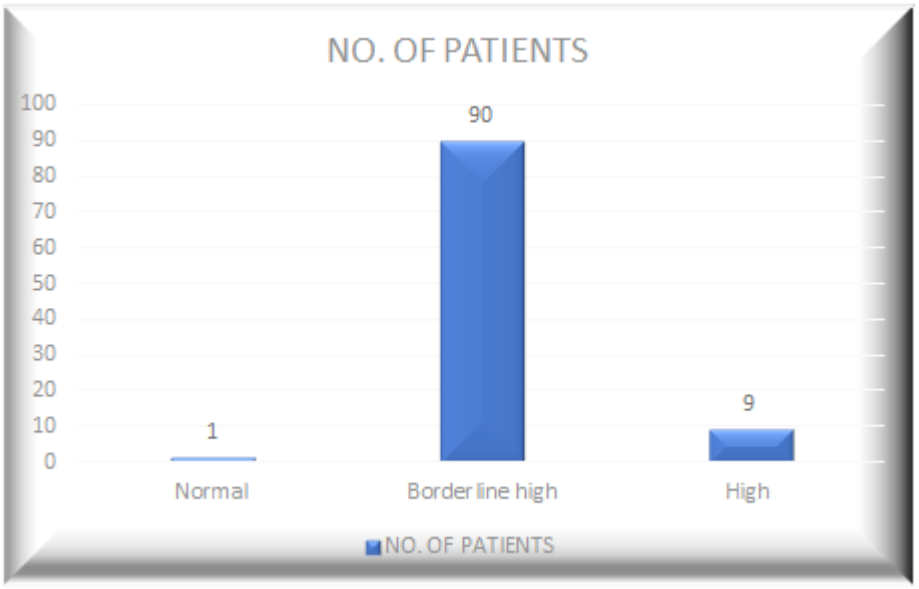
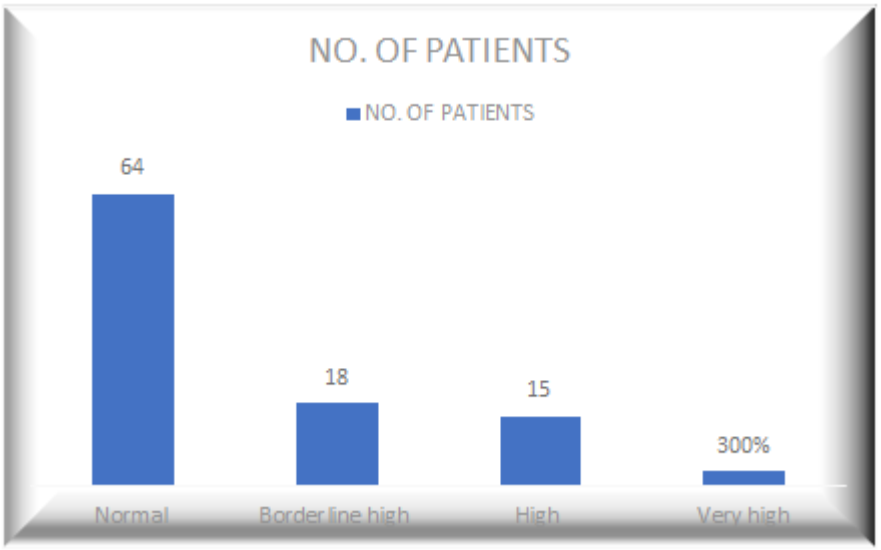


Table 6: Frequency table of triglycerides groups

IGLYCERIDES	. OF PATIENTS	EQUENCY
rmal		%
rderline high		%
gh		%
ry high	%	%

TRIGLYCERIDES WISE DISTRIBUTION:



DISCUSSION

The present prospective observational study sought to evaluate the efficacy, safety profile, and clinical implications of HMG-CoA reductase inhibitors (statins) in the real-world management of coronary artery disease (CAD). The findings reaffirm the integral role of statins, particularly Atorvastatin and Rosuvastatin, in managing dyslipidaemia and reducing cardiovascular risk in CAD patients.

Our study demonstrated that a significant majority (80%) of patients achieved normal low-density lipoprotein cholesterol (LDL-C) levels after one month of statin therapy. This outcome is consistent with previous clinical trials and real-world studies highlighting the potency of statins in lipid-lowering, including the Heart Protection Study and the TNT (Treating to New Targets) trial, both of which validated the role of intensive statin therapy in reducing LDL-C and improving cardiovascular outcomes.

Notably, Rosuvastatin and Atorvastatin—used either as monotherapy or in combination—were the predominant agentsprescribed. Both statins are considered high-potency and are widely recommended in current clinical guidelines for patients at high cardiovascular risk. The observed normalization of lipid parameters (LDL, HDL, triglycerides) further strengthens the case for their continued use in both primary and secondary prevention strategies.

While statins were well-tolerated overall, 73% of patients reported at least one adverse effect, with the most common being headache (28%), muscle pain (21%), and weakness (20%). These findings align with known statin-associated side effects documented in the literature. However, it is noteworthy that the majority of these effects were mild and self-limiting, reinforcing the safety profile of statins when used appropriately. A relatively low incidence of rare but concerning side effects such as myopathy and hepatotoxicity was observed, underscoring the importance of clinical monitoring during follow-up.

An interesting observation was the age-related trend in adverse effects, predominantly affecting individuals aged 41–80 years. This age group represents a population commonly prescribed statins, and age-related pharmacokinetic changes may contribute to the increased susceptibility to side effects. Tailoring dosages based on age, comorbid conditions, and pharmacogenomics could enhance patient outcomes and minimize intolerance.

The study also sheds light on an under-discussed yet clinically relevant aspect—patient-reported symptoms such as drowsiness, difficulty sleeping, and gastrointestinal discomfort. These symptoms, although not life-threatening, can impact adherence and overall quality of life. Strategies to enhance adherence include patient education, symptom monitoring, and dose adjustments, all of which align with the concept of precision medicine in cardiovascular therapeutics.

Furthermore, despite the overall lipid control, 20% of patients continued to exhibit elevated LDL-C levels. This residual hyperlipaemia may be attributed to suboptimal dosing, non-compliance, genetic dyslipidaemia (e.g., familial hypercholesterolemia), or lifestyle factors. It underscores the need for individualized care, including adjunctive therapies such as ezetimibe or PCSK9 inhibitors in statin-intolerant or refractory cases.

This study reinforces current guideline-directed medical therapy and highlights the utility of statins in achieving lipid control in CAD patients. The mild and manageable side-effect profile further supports their continued use in clinical practice. Given the emergence of precision medicine, incorporating pharmacogenomic profiling, risk stratification, and individualized therapeutic plans could further enhance efficacy and tolerability.

CONCLUSION

In conclusion, this study evaluated the clinical management of CAD patients treated with HMG-CoA reductase inhibitors, Atorvastatin, Rosuvastatin, Atorvastatin + Rosuvastatin, over a six-month period. Most patients experienced side effects, with muscle pain, weakness, headache, stomach pain being most common. Atorvastatin and Rosuvastatin was most frequently used across all age groups, while Atorvastatin + Rosuvastatin combination was primarily prescribed to patients with severe conditions. The study found that most of the patients experienced adverse effects. Despite some changes in lipid profile. Overall, both medications effectively reduce major cardiovascular events, with Atorvastatin, Rosuvastatin, Atorvastatin + Rosuvastatin being preferred for high-risk patients.

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