# SAFETY EFFECTIVENESS AND PATIENT –REPORTED OUTCOMES OF STATIN THERAPY:A 6-MONTHS PROSPECTIVE STUDY ON EDUCATIONAL INTERVENTIONS IN CAD MANAMGEMENT

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***Abstract :*** Atherosclerosis and modifiable risk factors are the main causes of coronary artery disease (CAD), which continues to be a major cause of morbidity and mortality worldwide. Over the course of six months, 180 statin-treated patients participated in this prospective interventional trial to assess the effects of pharmacist-led education on medication adherence, lipid profiles, and health-related quality of life. Participants were split up into groups for intervention and control. The results showed that the intervention group had a higher reduction in LDL (42.5%) and much better adherence (MARS ≈8.4) than the control group. There were a few mild adverse medication responses. The results demonstrate how systematic patient education can improve treatment outcomes and lower cardiovascular risk in people with CAD.

*IndexTerms* - Coronary Artery Disease, Atherosclerosis, Inflammatory, Myalgia, Palpitations, Headache, Myocardial Infarction, Hyperlipidemia, Atherosclerosis, Heart Stroke.

## INTRODUCTION

Global public health and healthcare economies face a significant challenge from coronary artery disease (CAD), which continues to be the world's leading cause of morbidity and mortality [1]. The Global Burden of Disease research continues to identify CAD as the leading cause of disability-adjusted life years (DALYs) and cardiovascular-related fatalities, accounting for more than 9 million deaths worldwide each year, despite notable progress in cardiovascular therapy [2]. Ageing populations, urbanisation, and a disproportionate burden of modifiable risk factors such as diabetes mellitus, hypertension, dyslipidemia, and sedentary lifestyles are the main causes of the rising prevalence of CAD [2, 3]. Understanding the intricate mechanisms underlying CAD, a major symptom of cardiovascular disease, is crucial for developing targeted prevention and treatment plans.

Atherosclerosis, a chronic, progressive, and highly inflammatory systemic disease that affects the epicardial coronary arteries, represents the core pathophysiology of CAD [4]. Endothelial dysfunction, which is frequently brought on by hemodynamic shear stress, oxidative stress, or circulating atherogenic lipoproteins, is the first step in the atherogenic process [4]. Low-density lipoprotein (LDL) cholesterol enters the artery intima and oxidises there when the endothelial barrier is breached. This oxidised LDL triggers a localised inflammatory response, attracting circulating monocytes that develop into macrophages [3, 4]. The oxidised lipids are forcefully consumed by these macrophages, which then develop into foam cells that build up to create the distinctive "fatty streaks" of early atherosclerosis.

This ongoing cycle of inflammation and lipid buildup eventually causes vascular smooth muscle cells to migrate and proliferate, resulting in the development of a fibrous cap over a lipid-rich necrotic core [5]. Depending on how these atherosclerotic plaques develop, CAD can present clinically in a wide range of ways. Myocardial blood flow is restricted by progressive luminal constriction, which results in a supply-demand mismatch that usually manifests as silent ischemia or stable angina [3]. However, the acute disruption of a "vulnerable" plaque is what causes the most catastrophic effects of CAD. The highly thrombogenic necrotic core is exposed to circulating blood when plaque ruptures or erodes, causing abrupt platelet activation and the coagulation cascade [5]. Acute coronary syndromes (ACS), such as myocardial infarction and sudden cardiac death, can ensue from the ensuing acute thrombus suddenly obstructing the coronary artery [4, 5].

Over the past few decades, the treatment of CAD has changed significantly, moving from only symptomatic alleviation to targeted revascularisation and comprehensive disease modification. In order to stabilise plaques and stop the course of the illness, current paradigms prioritise intensive risk factor management, antiplatelet therapy, and lipid-lowering medications (such as statins and PCSK9 inhibitors) [1, 5]. Additionally, the management of obstructive CAD has been transformed by improvements in coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCI), which have greatly decreased acute mortality rates [1]. Nevertheless, there is still a significant residual risk of recurrent cardiovascular events despite these therapies. It is crucial to keep researching the intricate inflammatory pathways, new biomarkers, and cutting-edge imaging techniques. In order to better understand the complex pathophysiology of coronary artery disease, this study will assess current diagnostic methods as well as new therapeutic options that could help lessen the illness's ongoing global impact.

## CORONARY ARTERY DISEASE:

### DEFINITION:

The narrowing or blockage of your coronary arteries, which provide your heart with oxygen-rich blood, is known as coronary artery disease (CAD). This occurs because the amount of blood that can reach your heart muscle is restricted over time by plaque (including cholesterol) accumulation in these arteries.The two primary types of coronary artery disease are: The chronic form is called stable ischemic heart disease. Over many years, your coronary arteries gradually narrow. Your heart gets less blood that is rich in oxygen over time. Even if you may experience certain symptoms, you can manage your disease daily.

Acute coronary syndrome: This is an emergency medical condition. Your coronary artery's plaque abruptly bursts, creating a blood clot that stops your heart from receiving blood. This sudden obstruction results in a heart attack. (6)

### *SIGNS AND SYMPTOMS:*

Chest pain and discomfort, or angina, is the most prevalent sign of coronary artery disease. When excessive plaque accumulates within arteries, it can constrict them and cause angina. Because narrowed arteries can obstruct blood flow to your heart muscle and the rest of your body, they can cause chest pain.

A heart attack is often the initial sign of CAD in many patients. Among the signs of a heart attack are

Angina, or discomfort or pain in the chest

Feelings such as weakness, dizziness, nausea, or a cold sweat

discomfort or pain in the shoulders or arms

Lack of breath

The heart muscle may deteriorate with time due to CAD. Heart failure, a dangerous illness where the heart is unable to pump blood as effectively as it should, could result from this. (7)

## HYPERLIPIDEMIA:

**DEFINITION:**AN OVERABUNDANCE OF LIPIDS OR FATS IN YOUR BLOOD IS KNOWN AS HYPERLIPIDAEMIA, OR HIGH CHOLESTEROL. BECAUSE IT MAKES IT MORE DIFFICULT FOR BLOOD TO PASS THROUGH YOUR ARTERIES, THIS MAY RAISE YOUR RISK OF HEART ATTACK AND STROKE. (8)

***TYPES AND CAUSES:***

**TYPES OF HYPERLIPIDAEMIA:**

Acquired hyperlipidaemia: This could result from specific actions you take (or don't take). In other cases, the illness may be caused by medications or other health conditions. Some people inherit it from their parents.
One kind of inherited hyperlipidaemia is familial hypercholesterolaemia. Instead of moving cholesterol out of the body, a mutation in the gene causes it to accumulate on the walls of the arteries.
Another kind of genetic hyperlipidaemia is familial hypertriglyceridemia. Triglyceride levels rise excessively as a result. [9]

**MYOCARDIAL INFARCTION:**

**DEFINITION:**Myocardial infarction (i.e., heart attack) is defined "pathologically as myocardial cell death caused by prolonged ischemia, through diminished cellular glycogen, and relaxed myofibrils and sarcolemmal disruption, which are the first ultrastructural changes and are followed by mitochondrial abnormalities." The clinical definition of Myocardial infarction is "the presence of acute myocardial injury as evidenced by abnormal cardiac biomarkers and acute myocardial ischemia. [10]

**TYPES OF MI:**

MI has been classified into five types based on the underlying cause:

- Type 1 (Spontaneous MI): It is related to the rupture or erosion of atherosclerotic plaques in the coronary arteries, which leads to the formation of a partial or complete thrombus in the lumen of the coronary arteries. This results in the reduced blood supply to the heart muscles and leads to MI. The patient may or may not have underlying obstructive CAD.
- Type 2 (MI secondary to an ischemic imbalance): It is MI secondary to an increase in myocardial oxygen demand and/or a decrease in myocardial oxygen supply (coronary endothelial dysfunction, coronary artery spasm, coronary artery embolism, tachyarrhythmias and bradyarrhythmias, anaemia, respiratory failure, hypertension, and hypotension), which is not associated with acute coronary atherothrombosis.
- Type 3 (MI resulting in death when cardiac biomarker values are unavailable): It is the unexpected death of the patient, in the presence of symptoms suggestive of MI and presumed ECG changes and ventricular arrhythmias, before the blood sample is taken for the estimation of the biomarkers and before the elevation of the biomarkers in the blood. It is also MI diagnosed at autopsy.
- Type 4a (MI related to percutaneous coronary intervention [PCI])
- Type 4b (MI related to coronary stent/stent scaffold thrombosis)
- Type 4c (MI related to coronary stent restenosis)
- Type 5 (MI related to coronary artery bypass grafting [CABG]): Identified up to and including 48 hours post-CABG. [11]

**ATHEROSCLEROSIS:**

**DEFINITION:**

Atherosclerosis is the accumulation of fatty deposits, or plaque, in your arteries. These deposits consist of fibrin, a blood-clotting agent, calcium, cellular waste materials, cholesterol, and fatty compounds. One kind of arteriosclerosis is atherosclerosis. Hardening of the arteries is known as arteriosclerosis.
The blood vessel's wall thickens as plaque accumulates. This causes the artery's channel to constrict, which lowers blood flow. The body receives less oxygen and other nutrients as a result. [12]

**HEART STROKE:**

**DEFINITION:**A stroke is an emergency medical condition in which the brain is deprived of blood flow and is usually caused by either a blockage in a blood vessel or the rupture of a blood vessel, causing it to bleed. When the brain is deprived of blood flow, brain cells die, and the results can be catastrophic and even fatal.

There are two types of strokes:

- Ischemic stroke is usually caused when a blood vessel is blocked by a blood clot, thereby depriving the brain of blood flow. This is the most common type of stroke and accounts for 80% of all strokes.

- A bleeding stroke is usually caused when a blood vessel ruptures and bleeds into the brain.

Another type of stroke is called a transient ischemic stroke, or 'mini-stroke.' This is usually caused when a blood vessel is blocked for a short time, and brain cells are deprived of blood flow for a short time. The brain cells recover, and there is no damage, but it increases the chances of having a stroke in the future. (13)

**STATIN THERAPY:**
Classification of statins:
HMG-CoA Reductase Inhibitors (Statins):

- Lovastatin
- Simvastatin
- Pravastatin
- Atorvastatin
- Rosuvastatin
- Pitavastatin

**MECHANISM OF ACTION:**

- Statins competitively inhibit HMG-CoA reductase, the enzyme that catalyses the rate-limiting step in cholesterol biosynthesis.
- The resultant reduction in hepatocyte cholesterol concentration triggers increased expression of hepatic LDL receptors, which clear LDL and LDL precursors from the circulation

**PHARMACOLOGY:**
Lovastatin, pravastatin, and simvastatin are derived from fungal fermentation. Fluvastatin, atorvastatin, and cerivastatin are entirely synthetic. Lovastatin, simvastatin, atorvastatin, and cerivastatin utilise the cytochrome P 450 (CYP) 3A4 pathway for metabolism or biotransformation. Fluvastatin metabolism occurs via CYP2C9, and pravastatin does not use the CYP pathway significantly. Pravastatin is extremely hydrophilic compared with other statins except for fluvastatin, which has intermediate physicochemical properties. This difference in hydrophilicity has not been demonstrated to have clinical significance.

**SIDE EFFECTS:**
Muscle aches, tenderness, or weakness.
Headaches.
Nausea.
Diarrhoea or constipation.
Indigestion or stomach discomfort.
Gas or flatulence.
Dizziness.
Sore throat.
Cold-like symptoms, such as a runny or stuffy nose.

**NEED OF STUDY:**

- To improve the patient's quality of life.

- To increase the patient's knowledge regarding statin therapy.
- To assess the patients' medication adherence towards the management of the disease.
- To increase the safety and efficacy of the drug.
- To reduce the morbidity and mortality rate in CVD.

**AIM:**

- Evaluation of statins therapy safety, effectiveness and medication adherence, HRQLC optimisation in CVD patients in a tertiary care hospital.

**OBJECTIVES:**

- To assess the impact of statins on patients' medication adherence behaviour.
- To evaluate the effectiveness of statin therapy in reducing the major events such as Myocardial infarction, Stroke, and Cardiovascular mortality.
- To assess the impact of statins on lipid profiles, such as:

1. Reduction in low-density lipoprotein cholesterol (LDL-C) level.
2. Increase in high-density lipoprotein cholesterol (HDL-C) level.
3. Reduction in triglyceride levels.
4. To evaluate the incidence of adverse effects (examples: muscle pain, liver enzyme elevation).
5. To decrease the mortality and morbidity.
6. To assess the impact of statin therapy on health care resources utilisation, including hospitalisation, op visits, and diagnostic tests.
7. To assess the impact of statin therapy on patient-reported outcomes such as HRQOL, symptom burden.

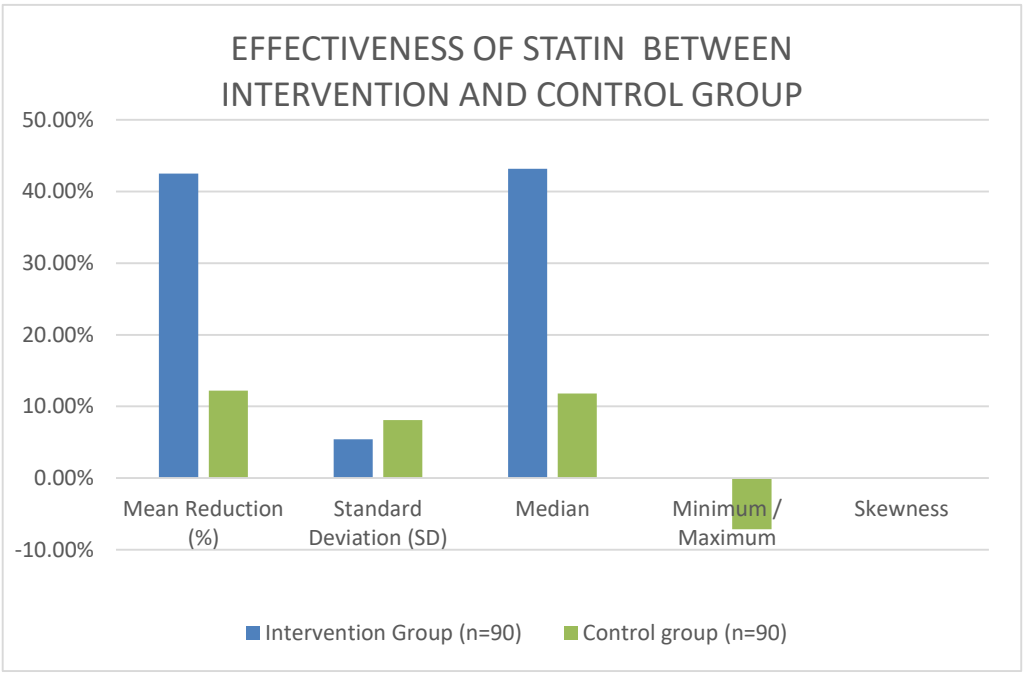
**RESEARCH METHODOLOGY:**

This study was a prospective, observational, and interventional study conducted over 6 months (September 2025 to February 2026) at Mamatha Hospital, a tertiary care teaching hospital. Patients aged ≥18 years with confirmed cardiovascular disease and on statin therapy were included after obtaining informed consent, while pregnant women and patients with severe comorbid conditions or cognitive impairment were excluded. A total of 180 patients were enrolled and randomised into two groups: control (routine care) and test (pharmacist-led education with patient information leaflets). Baseline data, including demographics, lipid profile, medication adherence (MARS), and health-related quality of life (HRQoL), were collected. Follow-ups were conducted at 30, 60, and 90 days to assess adherence, safety, and reinforce education in the test group. Final evaluation included lipid profile, MARS, and HRQOL assessment, with education provided to the control group at the end. Data were collected from case sheets, lab reports, and interviews, and analysed using Google Sheets and SPSS. Outcomes measured included medication adherence, quality of life, lipid profile changes, and adverse drug reactions using the Naranjo scale.

**RESULTS:**

**EFFECTIVENESS:**

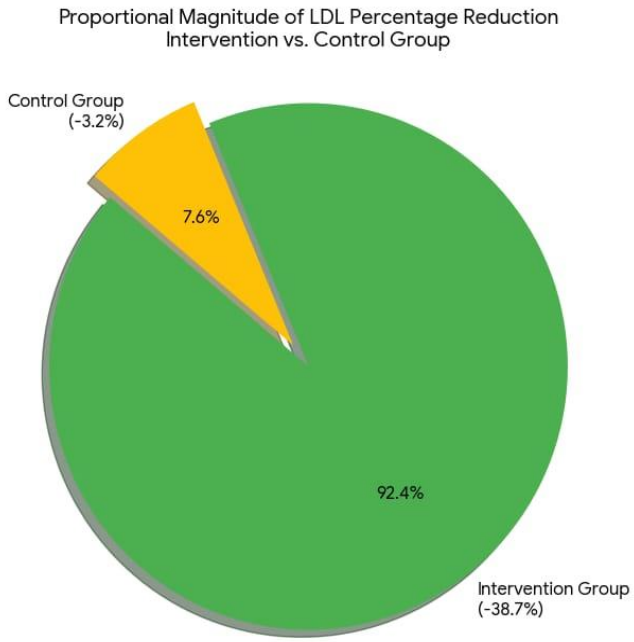
| STATISTICS | Intervention Group (n=90) | Control group (n=90) |
|---|---|---|
| Mean Reduction (%) | 42.5% | 12.2% |
| Standard Deviation (SD) | 5.4% | 8.1% |
| Median | 43.2% | 11.8% |
| Minimum / Maximum | 31.0% / 54.0% | -2.0%/ 28.0% |
| Skewness | -0.45(Slightly left) | 0.12(Near symmetric) |



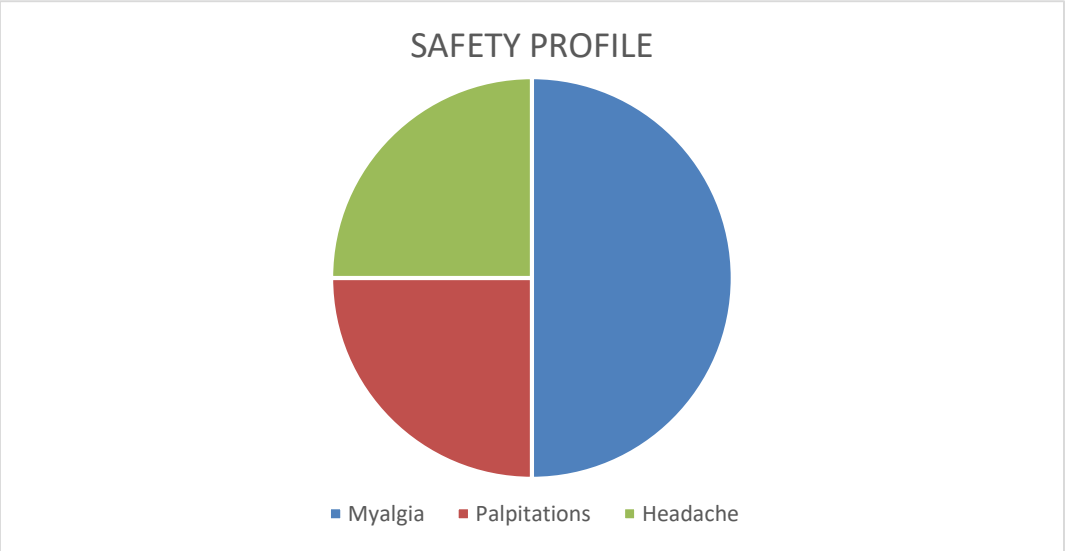

**INTERPRETATION:**

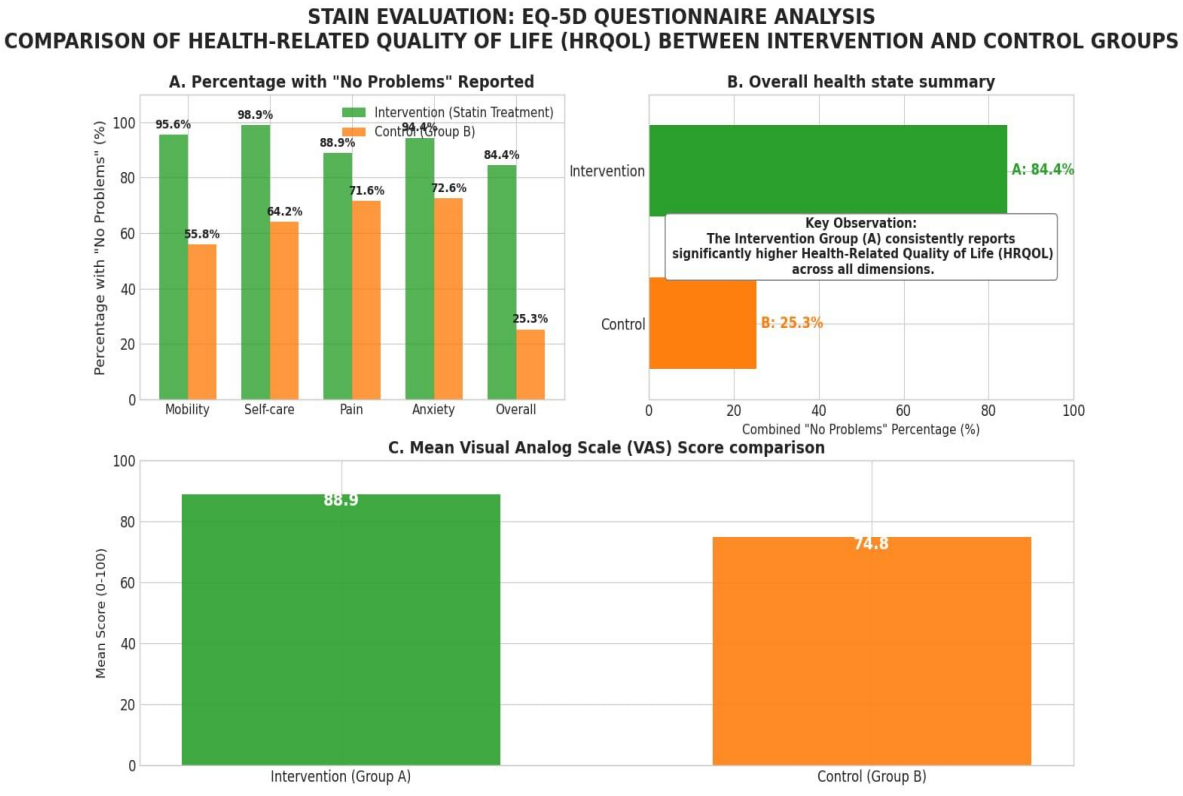
Standard care (Control) results in poor adherence (MARS approx. 4.5) and insufficient LDL reduction (12.2%). The intervention successfully stabilised adherence (MARS ≈8.4). This behavioural shift triggered a 30.3%absolute increase in LDL reduction(42.5% total), effectively reaching clinical targets for cardiovascular protection.

**SAFETY:**



| SUBJECT | Adverse drug event | No. Of sample | Naranjo score | Causality assessment | Hartwig's severity | Clinical outcome |
|---|---|---|---|---|---|---|
| 2026321524 | Myalgia | 2 | 6 | Probable | Grade -1 (mild) | Resolved without intervention |
| 2026385742 | Palpitations | 1 | 4 | Possible | Grade - 1(mild) | Transient; Self-limited |
| 2026985742 | Headache | 1 | 3 | Possible | Grade - 1(mild) | Spontaneous resolution |



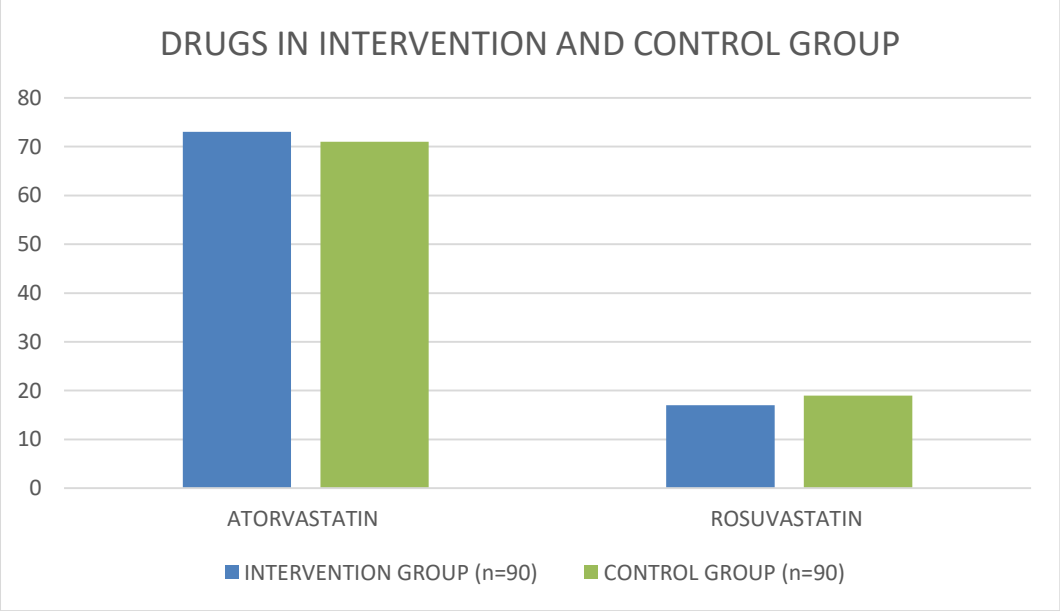



**HRQOL:**

A partial EQ-5D-5L questionnaire (mobility/walking, self-care/washing-dressing, pain/discomfort, anxiety/depression) and a VAS health assessment (0-100 scale) are included in the dataset, which includes responses from about 180 statin-treated cardiovascular patients. "This enables HRQOL assessment using VAS summary and EQ-5D index estimation.

***DRUG:***

| STATIN | INTERVENTION GROUP (n=90) | CONTROL GROUP (n=90) | Total (n=90) | Percentage |
|---|---|---|---|---|
| ATORVASTATIN | 73 | 71 | 144 | 80.0% |
| ROSUVASTATIN | 17 | 19 | 36 | 20.0% |
| Total | 90 | 90 | 180 | 100% |




INTERPRETATION: The comparability between the cohorts, the distribution of specific HMG-CoA reductase inhibitors (statins) was analysed. Atorvastatin was the primary agent in both groups, utilised by 81.1% of the intervention group and 78.9% of the control group. Rosuvastatin accounted for the remaining 18.9% and 21.1%, respectively. A Chi-square analysis ($\chi^2 = 0.139$, $p = 0.709$) confirmed no statistically significant difference in drug type distribution between the groups, ensuring that the variations in clinical outcomes (LDL reduction) were not due to the type of statin prescribed but rather the intervention provided.

**CONCLUSION:**

This study demonstrates that educational sessions conducted by pharmacists greatly improve clinical outcomes and medication adherence in individuals with coronary artery disease. Significant LDL reduction, reaching suggested therapeutic goals, and improved quality of life were all positively impacted by increased adherence. Only minor and self-limiting adverse medication reactions were noted, indicating that the intervention was safe. These findings highlight how important clinical pharmacists are to multidisciplinary care, especially when it comes to managing chronic illnesses. A more patient-centred approach to healthcare delivery can be supported by incorporating patient education into routine clinical practice, which can successfully lower residual cardiovascular risk and enhance long-term prognosis in CAD patients.

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