Lipid profile in coronary artery disease under the treatment of lipid lowering agents

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***Abstract*- Globally, Coronary artery disease is one of the most common health problems. the lipid profile is one of the major risk factors for worsening the condition. This study evaluated the lipid profile of patients with Coronary artery disease who were being treated with lipid-lowering agents with the aim of reducing the risk of heart attacks and improving their health outcomes. I have investigated that Mono therapy of Atorvastatin 40mg effectively reduces LDL-C levels with fewer side effects. The complete blood analysis of 74 patients was monitored with the standard reference range. Among 74 patients, 18.5 % (n= 25) of the subjects have shown Low-density lipids, 14.8% (n=20) are total cholesterol, 14.6% (n=19) are the patients with triglycerides & 2.2 % (n=3) with elevated VLDL & 5.1% (n=7) shows the status of the HDL cholesterol. The study procedures were conducted by the ethical principles of the Declaration of Helsinki and its amendments with the CDSCO recommendations, the Indian Council of Medical Research, and GCP guidelines.**

***Index Terms*- Coronary Artery Disease, Cholesterol, Cardiovascular, LDL, HDL.**

I. INTRODUCTION

Coronary Artery Disease (CAD) is the most common form of Heart disease worldwide. Coronary artery disease develops when the major blood vessels that supply the heart with blood, oxygen and nutrients (coronary arteries) become damaged, diseased, hardened or narrowed due to build up of cholesterol and other materials called plaque. This build up/plaque is called atherosclerosis.When plaque builds up, they narrow the coronary arteries, decreasing blood flow to the heart. Eventually, the decreased blood flow may cause chest pain (angina), shortness of breath, or other signs and symptoms of coronary artery disease. A complete blockage can cause a heart attack.[1],[5],[9]

Total plasma lipid is 400-600mg/dl. Depending on the density or on the electrophoretic mobility, the lipoprotein in plasma are classified into 5 major types, Chylomicrons, VLDL(Very Low Density Lipoproteins), LDL (Low Density Lipoproteins), HDL (High Density Lipoproteins), Triglycerides.[10],[15].

Chylomicrons are the transport form of dietary triglycerides from intestines to the adipose tissue for storage; and to muscle or heart for their energy needs.

VLDL carries triglycerides (endogenous triglycerides) from liver to peripheral tissues for energy needs.[22],[23].

About 75% of the plasma cholesterol is incorporated into the LDL particles. LDL transports cholesterol from liver to the peripheral tissues. The cholesterol thus liberated in the cell has three major fates: (i)It is used for the synthesis of other steroids like steroid hormones. (ii) Cholesterol may be incorporated into the membranes. (iii)Cholesterol may be esterified to a MUFA by acyl cholesterol acyl transferase (ACAT) for storage. The cellular content of cholesterol regulates further endogenous synthesis of cholesterol by regulating HMG CoA reductase.[10],[15]

HDL is the main transport form of cholesterol from peripheral tissue to liver, which is later excreted through bile. This is called reverse cholesterol transport by HDL. The only excretory route of cholesterol from the body is the bile. Excretion of cholesterol needs prior esterification with PUFA. Thus PUFA will help in lowering of cholesterol in the body, and so PUFA is anti-atherogenic. [10],[15]

Triglyceride is also known as non esterified fatty acids (NEFA). It is complexed with albumin in plasma. The FFA is derived from lipolysis of triglyceride stored in adipose tissue by hormone sensitive lipase. Free fatty acids may be long chain saturated or unsaturated fatty acids. The FFA molecules are transported to heart, skeletal muscle, liver and other soft tissues. The free fatty acids are either oxidised to supply energy or incorporated into tissue lipids by esterification. In the tissue cells, FFA-albumin complex is dissociated, FFA binds with a fatty acid transport protein. It is a co-transporter with sodium. After entry into the cell, the FFA is bound to fatty acid binding protein. The half-life of free fatty acids in plasma is very short; only 1-2 minutes. During starvation, about 40-50% energy requirement of the body is met by oxidation of FFA. Blood level of FFA is very low in the fully fedncondition, high in the starved state, and very high in uncontrolled diabetes mellitus.[22],[23].

II. METHODOLOGY

2.1 Study Site And Study Design:

This was a prospective open observational study of patients referred to the Department of cardiology at Apollo hospitals, Kakinada for patients with coronary artery disease under the treatment from February 2019 to July 2019. The study was approved by the Institutional Ethics Committee. Each patient was provided with a written informed consent and patient acceptance was obtained.

The study procedures were conducted in accordance with the ethical principles of the declaration of Helsinki and its amendments with the CDSCO recommendations, Indian Council of Medical Research and GCP guidelines.

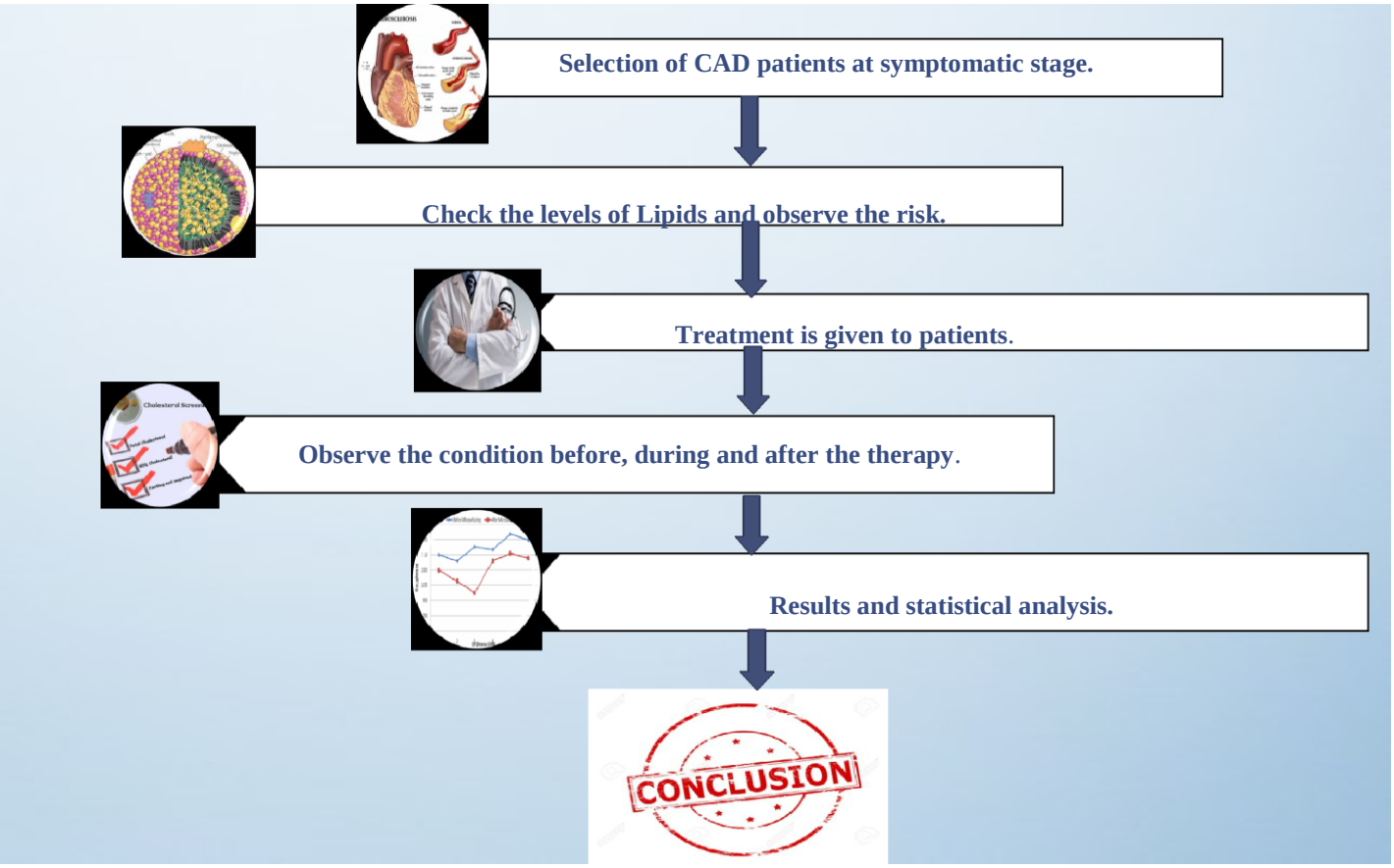
2.2 Study Criteria:

2.2.1Inclusion criteria- CAD patients, Patients with diabetes, Both male and female patients above 25 years.

2.2.2 Exclusion criteria- Pregnant women and patients having renal complications.

2.2.3 Patient selection and procedures: To evaluate the lipid profile of the patients with coronary artery disease. 100 patients with the initial diagnosis of CAD were selected for the study. On examination of signs and other prevailing symptoms, CAD was confirmed. All patients involved to meet the definition of CAD in accordance with the criteria

2.2.4 Study Design



The entire patient profiles were entered in a standard medical history questionnaire with an emphasis on components like: Lipid profile, Diabetes mellitus, Hypertension and Co morbidities like: COPD, asthma, surgical, medical and family history.

2.3 Procedures And Definitions: All of the patients underwent a complete screening panel, including Physical, Biochemical examinations.

2.3.1Physical Examinations: Patients presenting with symptoms of chest pain are subjected to heart check profile which includes TMT/ Blood glucose, BP, Lipid profile etc.

2.3.2 Biochemical Examination: Blood samples were collected and lipids were evaluated.

Table 2.3.2 Normal values

Analyte	**Normal Value**
Total plasma lipid	400-600mg/dl
Total Cholesterol	150-200mg/dl
HDL (Male)	30-60mg/dl
HDL (Female)	35-75mg/dl
LDL (30-39 yrs)	80-175mg/dl
Triglycerides (Male)	50-200mg/dl
Triglycerides (Female)	40-150mg/dl
Phospholipids	150-200mg/dl
Free fattyacids	10-20mg/dl

2.3.3 Metabolic Evaluation: Lipid Profile: Total cholesterol (reference range from 150 to 234 mg/dl), low-density lipoprotein (LDL) (reference range from 60 to 190 mg/dl), high-density lipoprotein (HDL) (reference range from 35 to 60 mg/dl), and triglycerides (TG) (reference range from 150 to 500 mg/dl) were regularly measured after an overnight fasting period of 12 h, using routine clinical chemistry methods, and documented

2.4 Statistical Analysis: The analyses, which were done using SAS 6.1.2, addressed the change in lipid and apolipoprotein variables and lipoprotein sub fractions before and after atorvastatin treatment. Before analysis, triglyceride, VLDL-C, VLDL-TG, and IDL-TG values were logarithmically transformed. Untransformed concentrations are reported in the tables. Significance was assessed at the 5% level of probability.

III. RESULTS

This was a prospective open observational study of patient referred to the Department of Cardiology at Apollo hospitals, Kakinada for patients with coronary artery disease under the treatment from February 2019 to July 2019. The study was approved by the Institutional Ethics Committee. Each patient was provided with a written informed consent and patient acceptance was obtained. The study procedures were conducted in accordance with the ethical principles of the declaration of Helsinki and its amendments with the CDSCO recommendations, Indian Council of Medical Research and GCP guidelines.

Table 3.1 Baseline demographic characteristics.

S. no	**List**	**Total n=79**
1.	**Demographics:** Total	79
	Age	48.3
	Sex	40.09% (51) - Male 20.12% (28) - Female
	Lipid profile	58.4%(74)
2	**History :** CAD	39% (50)
	HTN	56.8%(72)
	DM	49.7%(63)
	Smoking and alcohol consumption	15%(20)

3.1. Risk Factor

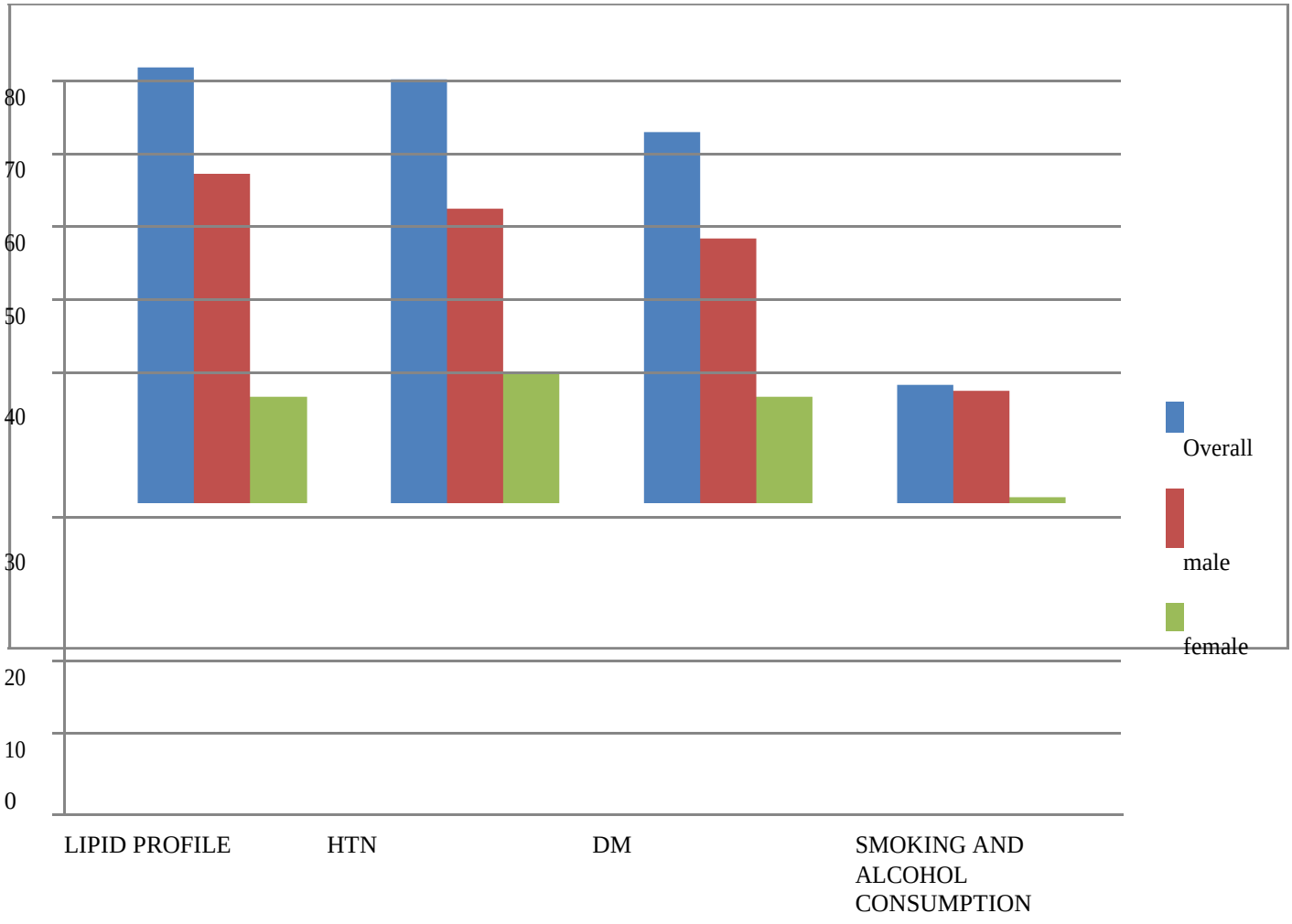


Figure 3.1: No. of patients at risk with Lipid profile, Hypertension, Diabetes, Smoking and Alcohol Consumption

Table 3.2 Lipid Profile Data Set

Lipds	Subjects With Normal Values	Percentage
Total Cholesterol	20	14.08%
Triglycerides	19	14.6%
HDL	7	5.18%
LDL	25	18.5%
VLDL	3	2.2%
Total	74	

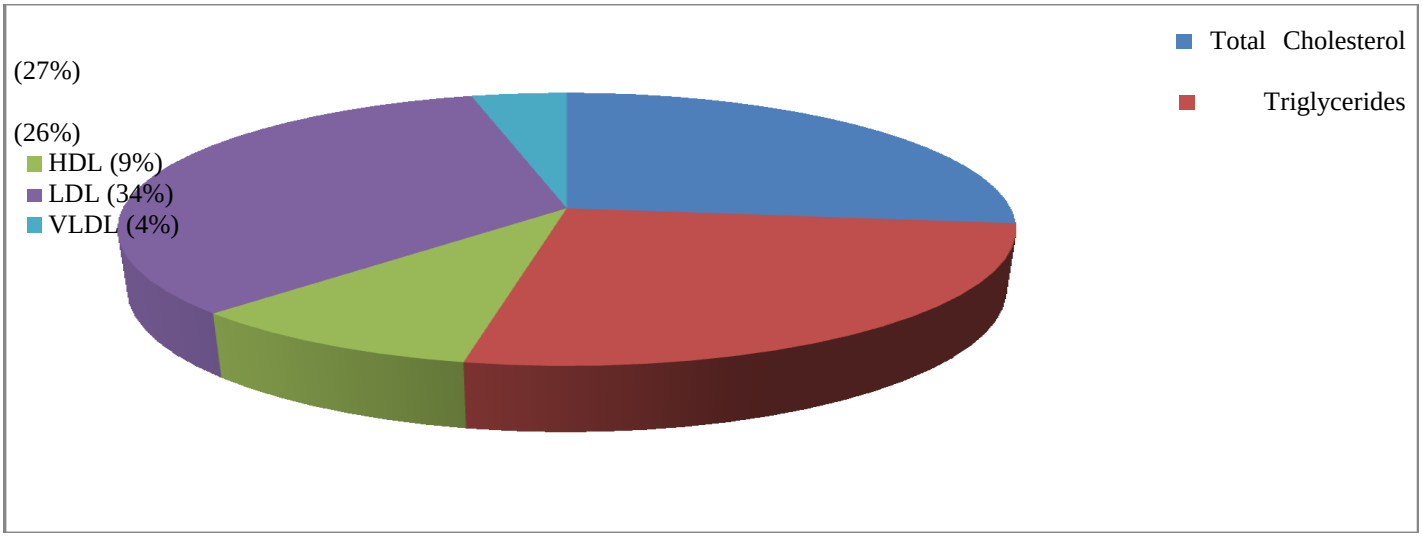


Fig 3.2 Lipid Profile Data Set

3.3 Lipid profile in patients with CAD and Percentage of Abnormality in Lipid profile in patients with CAD:

The complete blood analysis of 74 patients was monitored with the standard reference range. Among 74 patients, 18.5 % (n=25) of the subjects have shown Low density lipids, 14.8% (n=20) are total cholesterol, 14.6% (n=19) are the patients with triglycerides & 2.2 % (n=3) with elevated VLDL & 5.1% (n=7) shows the status of the HDL cholesterol.

From the above data set it is clearly evident that 18.5% of the subjects with LDL cholesterol are having risk of CAD.

Fig 3.3 Efficacy of monotherapy of Atorvastatin 40mg

Fig 3.3.1 Total Cholesterol

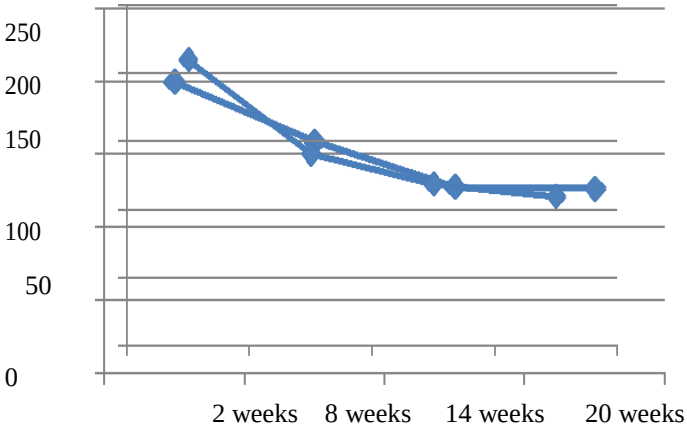


Fig 3.3.2 Triglycerides

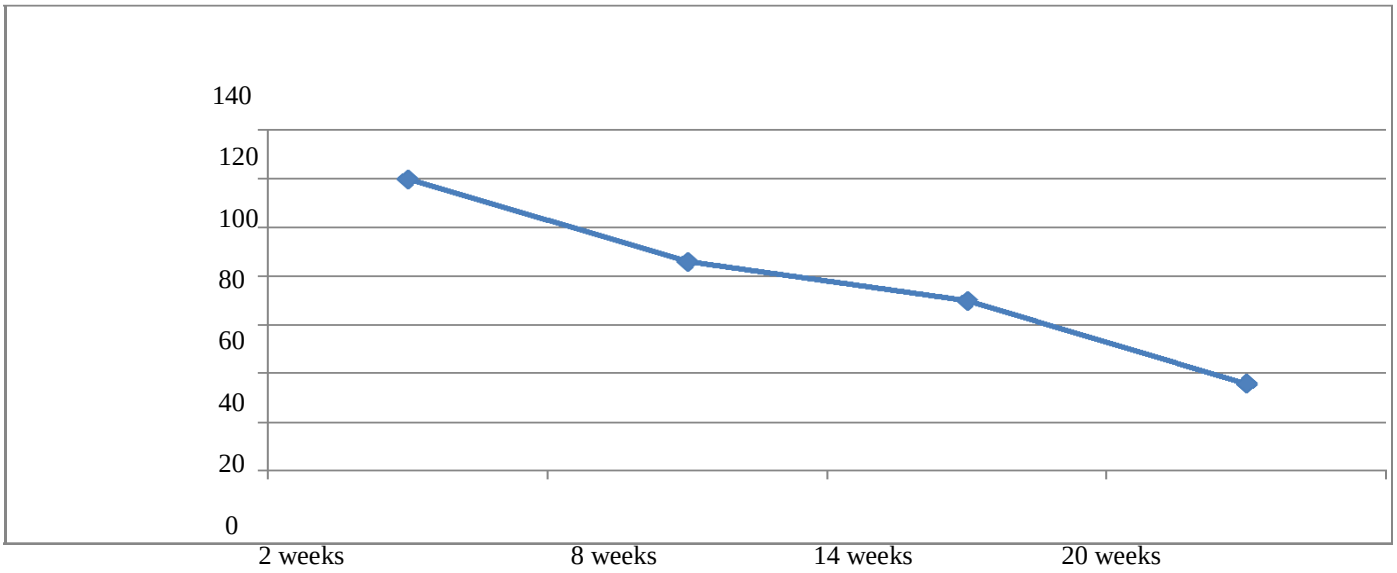


Fig 3.3.3 – LDL C

Table 3.3Average no.of patients who show significant decrease with monotherapy of Atorvastatin 40mg.

S. no	Lipid profile.	No. of subjects out of 74	P value
1	Total cholesterol	29/74	0.39
2	Triglycerides	32/74	0.4
3	LDL-C	56/74	0.75

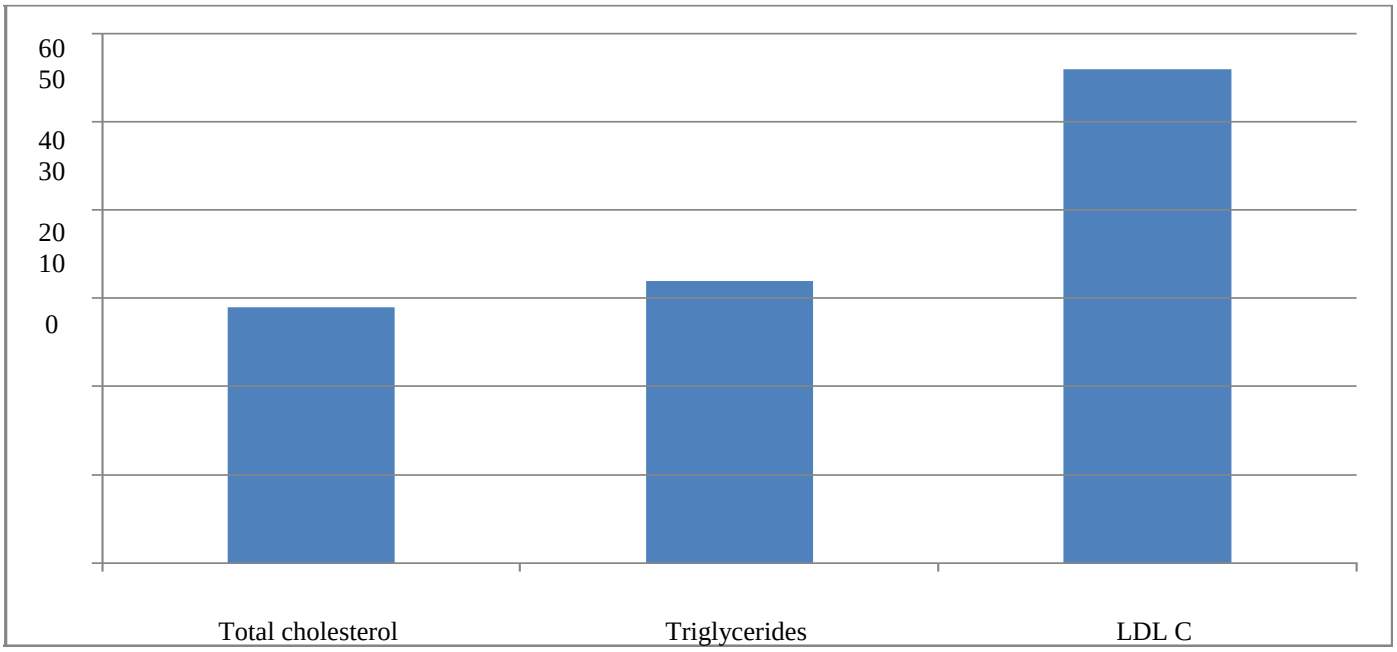


Fig 3.4 Average no.of patients who show significant decrease with monotherapy of Atorvastatin 40mg. Total cholesterol (p=0.39), Triglycerides (p=0.4), LDL-C (p=0.75).

Table 3.4Difference calculated in the decrease of Total cholesterol, Triglycerides and LDL-C between 1st month and 6th month.

S. no	**Lipids**	**Difference**	**P-value**
1.	Total cholesterol	23	0.31
2.	Triglycerides	34	0.46
3.	LDL-C	62	0.84

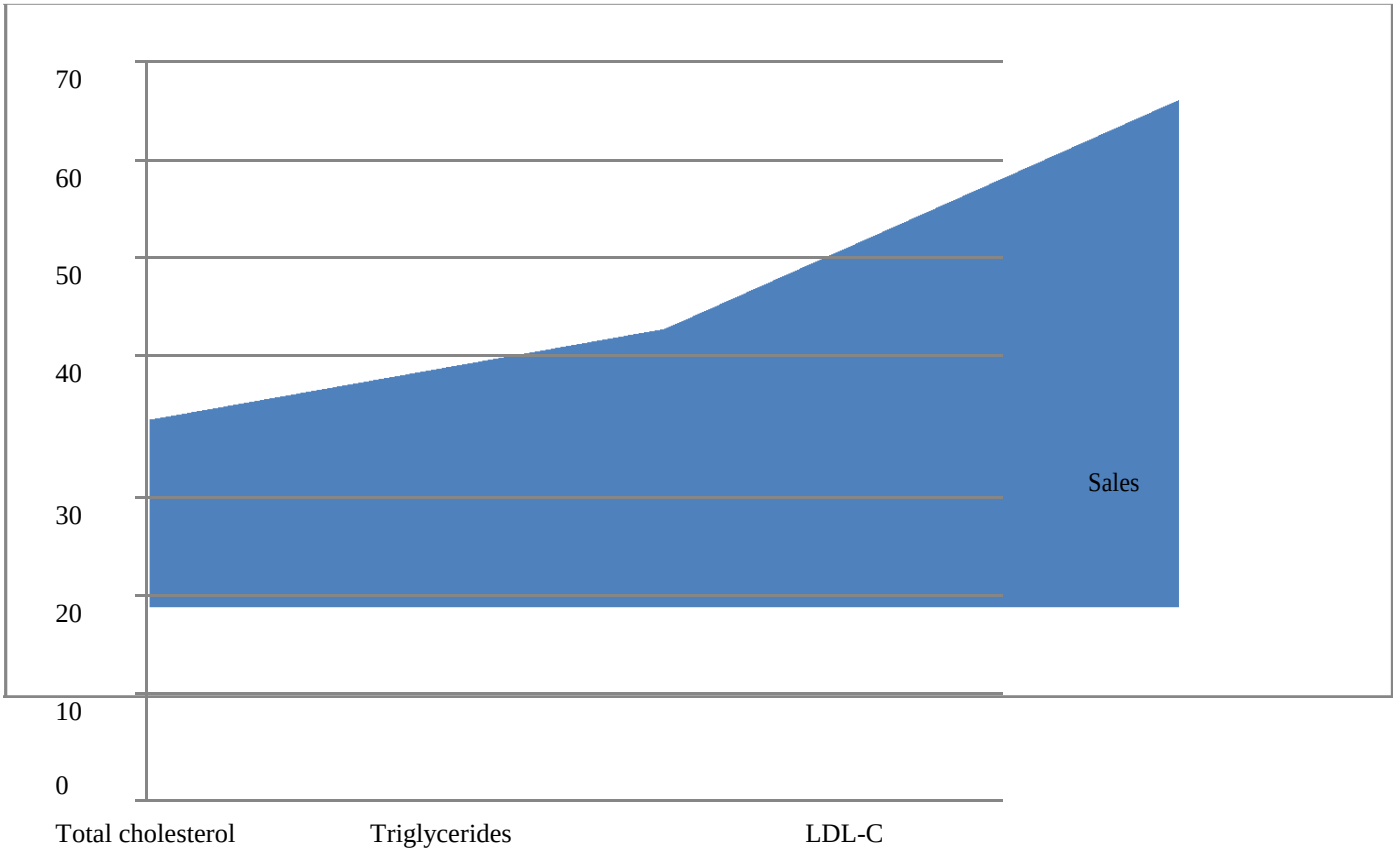


Fig 3.5 Difference calculated in the decrease of Total cholesterol, Triglycerides and LDL-C between 1st month and 6th month.

IV. DISCUSSION

Coronary Artery Disease is a major heart occurring throughout the world. There are several risk factors that prevailed in the severity of heart disease. Among the risk factors, the lipid profile provides a new insight into atherosclerosis which is the major cause of CAD. Among the risk factors Lipid profile (n=74) is occupying the 1st place followed by Hypertension (n=72). Therefore observing the lipid profile along with its depletion with the correct choice of lipid-lowering agent is included in my study. My study indicates that patients receiving the monotherapy of atorvastatin 40mg have a significant decrease in the lipid profile, especially LDL-Cholesterol followed by total cholesterol and triglycerides. This interesting outcome could guide clinicians to select the proper statin in the treatment of higher levels of bad cholesterol i.e. LDL-C. LDL-C which is considered bad cholesterol would accumulate in the coronary arteries and cause the artery to be damaged, which when corrected with the right lipid-lowering agent would make the condition better.

V. CONCLUSION

The study is to observe the lipid profile in patients with CAD before and after the therapy and it has demonstrated that total cholesterol, Triglycerides, and LDL-C have shown a steep decrease, among which LDL-C is reduced more rapidly. I have explored that Monotherapy of Atorvastatin 40mg effectively reduces LDL-C levels with fewer side effects. The study has certain limitations like a small sample size. I suggest for the next study that there would be a large no. of participants with all types of cardiovascular complications.

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