CODEN [USA]: IAJPBB ISSN : 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187
https://doi.org/10.5281/zenodo.7968900

Available online at: http://www.iajps.com *Research Article*

EXPLORING THE BENEFITS OF CLINICAL PHARMACIST'S COUNSELING IN ENHANCING THE MEDICATION COMPLIANCE AND LOW-DENSITY LIPOPROTEIN MANAGEMENT OF HYPERCHOLESTEROLEMIC PATIENTS

Dr. Pavan Kumar Yanamadala*[1], Lalitha Sanjana Nallaparaju[2], Lakshmi Srilekha Danturthi[3], Borusu Balavendra Srinivas[4]

*[1]Aditya Pharmacy College, Surampalem-533437, East Godavari District, Andhra Pradesh, India
[2]Aditya Pharmacy College, Surampalem-533437, East Godavari District, Andhra Pradesh, India
[3]GIET School of Pharmacy, Rajamahendravaram, East Godavari District, Andhra Pradesh, India
[4]Aditya Pharmacy College, Surampalem-533437, East Godavari District, Andhra Pradesh, India

Abstract:

Low-Density Lipoprotein Cholesterol reduction reduces the risk of recurrent Myocardial Infarction and death in Coronary Artery Disease patients and in unhealthy individuals. Reduced levels of low-density lipoprotein also inhibit the development of coronary atherosclerosis. By preparing and encouraging patients to adhere to their pharmacotherapeutic regimens, dietary modifications, and management plans, clinical pharmacists can help to ensure that pharmacotherapy has beneficial results. It is clear that the initiatives to increase adherence and ongoing lipid-lowering medication use in high-risk patients are required. Long-term face-to-face counseling is necessary for effective care but may not be practical for many patients. A different strategy is to follow up by telephone. So, in high-risk hypercholesterolemic patients, we evaluated the effect of personalized telephone follow-up on the rate of compliance. As shown in this study, compared to patients without clinical pharmacist management of Dyslipidemia, patients with multiple risk factors who are treated by an interdisciplinary medical team that includes clinical pharmacists in lipid management experienced greater reductions in Low-Density Lipoprotein levels. Improved intermediate results in achieving lipid goals were obtained from the Clinical Pharmacist's active participation in lipid management for all patients with elevated Low-Density Lipoprotein Levels. Medication Adherence was also assessed using the Morisky Medication Adherence Scale and the mean scores for the Intervention group showed an increased rate of medication adherence when compared to the control group without intervention. The mean score of 5.15 got increased to 6.85 in the Intervention group after 3 months whereas the mean score of 5.125 got a slight increase to 5.225 in the control group. These interim results could eventually lead to fewer long-term cardiovascular events and better patient quality of life.

Keywords: *Low-Density Lipoprotein Management, Clinical Pharmacist Interventions, Medication Adherence, Treatment Compliance, Coronary Artery Disease, Hypercholesterolemia*

Corresponding author:

Dr. Pavan Kumar Yanamadala,

Assistant Professor, Department of Pharmacy Practice,
Aditya Pharmacy College, Suramapalem-533437,
East Godavari District, Andhra Pradesh, India
Email- pavan.yanamadala@gmail.com

QR code

Please cite this article in press Pavan Kumar Yanamadala et al, ***Exploring The Benefits Of Clinical Pharmacist's Counselling In Enhancing The Medication Compliance And Low-Density Lipoprotein Management Of Hypercholesterolemic Patients.,*** *Indo Am. J. P. Sci, 2023; 10 (05).*

INTRODUCTION:

Lowering low-density lipoprotein (LDL) cholesterol reduces the risk of recurrent myocardial infarction and death in healthy subjects and patients with coronary artery disease.[1-7] Lowering LDL levels also slows the progression of coronary atherosclerosis. [8, 9]

Although the continuation of pharmacotherapeutic treatment is essential for patients with Hyperlipidemia, many of them do not adhere to the prescribed treatment regimen because Hyperlipidemia is a painless condition and is usually unnoticed by the patient. A recent cohort study further showed that primary care dropout rates were higher than in clinical trials, suggesting that non-adherence to lipid-lowering medications is a major problem in routine clinical practice. Advise the patients to prepare and encourage them to adhere to the treatment and follow-up plans. [10]

Pharmacists can help improve pharmacotherapy outcomes by teaching and counseling patients to help them prepare for and stick to their pharmacotherapeutic regimens and monitoring plans. Numerous strategies for decreasing LDL-C have undergone research. These include lipid apheresis, intestinal bypass surgery, pharmaceutical therapy, and lifestyle changes. The evidence supporting pharmacological and lifestyle therapies, as well as their impact on cholesterol control recommendations, are the main topics covered below.

Studies have found that clinical pharmacist-managed lipid clinics improved these outcomes.[11, 12] It is evident that measures to increase lipid-lowering drug compliance and long-term use are required, particularly in high-risk patients. Effective management necessitates long-term face-to-face counseling, which may be prohibitively expensive for large numbers of patients or those living in remote places.

Telephone follow-up is an additional method for increasing compliance and improving outcomes. As a result, we investigated the effect of personalized phone follow-up on compliance in high-risk, hypercholesterolemic patients on combination pharmacological therapy.

Higher population LDL-C levels are a result of societal changes brought on mostly by agricultural and industrial growth. Evidence from hunter-gatherers has shown that these populations often had LDL-C values between 50 and 75 mg/dL. Even among those who live up to eight decades, atherosclerosis is absent in these populations. Additionally, LDL values in healthy, wild, adult primates range from 40 to 80 mg/dL.[13, 14] In contrast, Westernised societies currently view a range of 100 to 160 mg/dL as normal.[15]

There are other dietary adjustments that can lower cholesterol. According to a meta-analysis of 87 well-controlled studies, eating 2 to 10 grams of dietary soluble fiber daily lowers LDL Cholesterol by 2.2 mg/dL.[16] Every 2.15 g of daily phytosterol ingestion lowers LDL Cholesterol by 13 mg/dL.[17] Daily ingestion of 67g of nuts reduces LDL Cholesterol by 10.2 mg/dL and that of soy isoflavones by 5 mg/dL.[18, 19] Additionally, it has been demonstrated that the quantity of small LDL particles is positively connected with dietary cholesterol intake, high-carbohydrate diets (and particularly diets with a high glycemic index), and trans-fatty acid (TFA) consumption. [20, 21] TFA usage has decreased over the past three decades as a result of attempts to banish industrial TFA from foods, despite the fact that it is associated with noticeably increased LDL Cholesterol levels.[22]

Beyond specific foods, it has been demonstrated that comprehensive diets like the Mediterranean diet, which predominantly consists of fruits, vegetables, legumes, grains, nuts, and olive oil, can reduce LDL-C by 10% after 5 weeks.[23] According to a recent study, persons who followed the Mediterranean diet for at least 10 years had a 47% lower risk of developing heart disease than similar adults who did not.[24] The stricter Ornish diet has been demonstrated to reduce LDL-C by 37%, although it is extremely difficult to follow.[25] Exercise training does not significantly lower LDL-C levels in the absence of weight loss.[26-29] Randomized studies show, however, that exercise reduces the quantity of tiny Low-Density Lipoprotein particles.[30-32] Therefore, a change from smaller, more atherogenic particles to fewer, larger particles may help to partially account for the decreased CV risk linked to physical activity[33]. An improved diet and exercise plan often reduces LDL-C by 10-15%.[34]

The aim of this study is to investigate the effects of individualized telephone follow-up on drug therapy compliance rates and the changes in the Low-Density Lipoprotein levels in hypercholesterolemic individuals.

Objectives:

1) To research the demographics of patients with Hyperlipidemia
2) To investigate the effects of Hyperlipidemic individuals' medication non-adherence

3) To investigate the effects of individualized telephone follow-up on the rate of compliance decline and cholesterol level in patients with Hyperlipidemia.

METHODOLOGY:

Study Site: Subjects -are enrolled from the Outpatient department of -Trust Hospital, Kakinada, East Godavari District of Andhra Pradesh, India.

Study Population: The sample size is 80 subjects who had been diagnosed with Dyslipidemias and were being treated with Statins and other Lipid Lowering agents. They were randomly assigned to two groups of 40 each. (Test Group and Control Group)

Study Duration: Data Collection of Baseline levels and Final values of LDL Profile tests took 3 and half months. Data and Statistical Analysis took one month. Data was evaluated and assessed through Mathematical (Microsoft Excel) and Statistical Calculations (Z-test)

80 Subjects were chosen from Tertiary care hospital-affiliated free-standing outpatient clinics. Patients who had Congenital Heart Defects, and were between the ages of 30 and 80 years, taking at least aspirin or other acceptable treatments (Clopidogrel, Ticlopidine, Aspirin, and Ticagrelor), and had received refill prescriptions for lipid-altering drugs (HMG-CoA reductase inhibitors, niacin, and bile acid sequestrants) during the preceding six months were eligible for the study. Patients with baseline fasting Low-Density Lipoprotein levels exceeding 130 mg/dl were included in the study. They had to be able to communicate in Telugu, speak it, understand it, and have a landline or cell phone at home. Each participant signed a consent form after receiving full information. When the prescriptions were written, each participant receives intensive counseling on the proper utilization of the medications.

The study's primary endpoints were the proportion of patients who met their LDL goal of less than 100 mg/dl + 5% (while excluding those with triglycerides greater than 400 mg/dl); a goal LDL of less than 105 mg/dl was chosen because the laboratory assay has a margin of error of + 5% and we wanted to leave some room for clinical judgment on the part of the healthcare professionals. A list of random numbers produced by a computer was used to create a randomization schedule. After satisfying the inclusion requirements and consenting to participate in the study at their initial follow-up appointment, subjects were randomly assigned to either a treatment group or a control group.

The lipid management program, which was overseen by a Trainee Clinical pharmacist, was implemented for subjects who had been included in the treatment group. Subjects in the control group were notified of their cholesterol readings and advised to get in touch with their doctor for additional follow-up. At the study's baseline, lipid profiles were assessed for the first time and three months after it began for the final reading. For three months, trainee clinical pharmacists called the patients in the intervention group at their homes once for 2 days through the contact number provided. An emphasis was made on the value of therapy in lowering the risk of subsequent cardiac episodes during the telephonic conversation. Patients were enquired about probable side effects, general health, and if necessary, specific causes of non-compliance. Information about the dosage, frequency, and indication of lipid-lowering medications is provided as part of the counseling process. There is a brief discussion of dietary guidance also. A thorough assessment of the related study's literature served as the basis for the questionnaire that was used to collect the data. The questionnaire asked about the demographics and clinical characteristics of the patient, including details like Gender, age, education, income, medical history, and co-morbidities. The process employed in this study was divided into three parts: the first part was to collect the socio-demographic, clinical, and medication data from patient's medical records; the second part was a test of medication adherence; and the third and final component was a survey of treatment satisfaction. The eight-item Morisky Medication Adherence Scale (MMAS-8), which has been validated, was used to assess medication adherence. The MMAS-8 is an 8-item questionnaire with 7 yes/no questions and a 5-point Likert scale as the final question. According to the MMAS scoring method, adherence was classified as having a high level (=8), a medium level (6 to 8), and a poor level (6). Non-adherent patients were those who had a low or moderate rate of adherence. Each item evaluates a distinct method of taking medication.

RESULTS:

TABLE 1 GENDER WISE DISTRIBUTION OF SUBJECTS

Gender	Intervention (Test Group) (n=40)	Routine Care (Control Group) (n=40)	Total Number of Subjects (n=80)
Male	31 (77.5%)	28 (70%)	59 (73.75%)
Female	09 (22.5%)	12 (30%)	21 (26.25%)

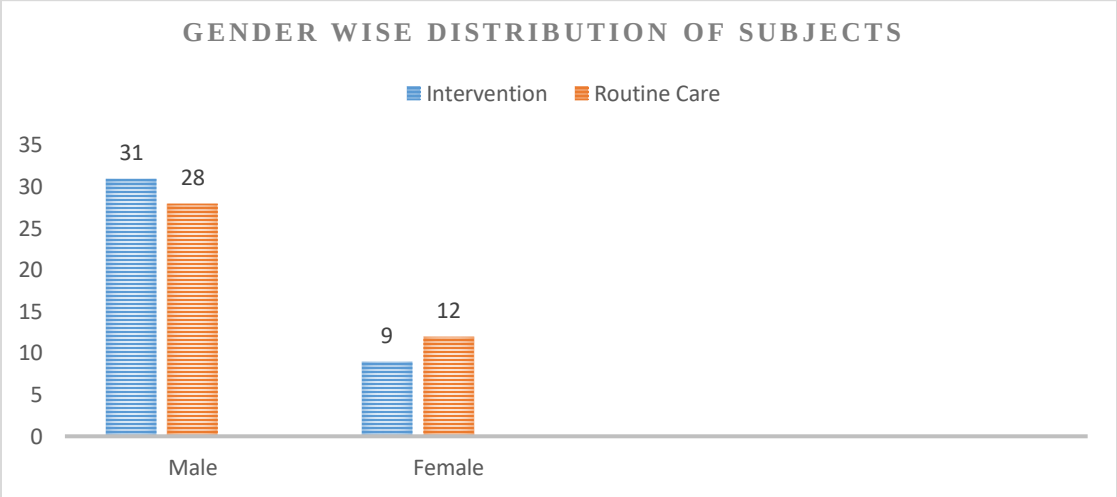

FIGURE 1 GENDER WISE DISTRIBUTION OF SUBJECTS

TABLE 2 AGE WISE DISTRIBUTION OF SUBJECTS

Age in Years	Intervention (n=40)	Routine Care (n=40)
< 50 Years	5 (12.5%)	6 (15%)
> .50 Years	35 (87.5%)	34 (85%)

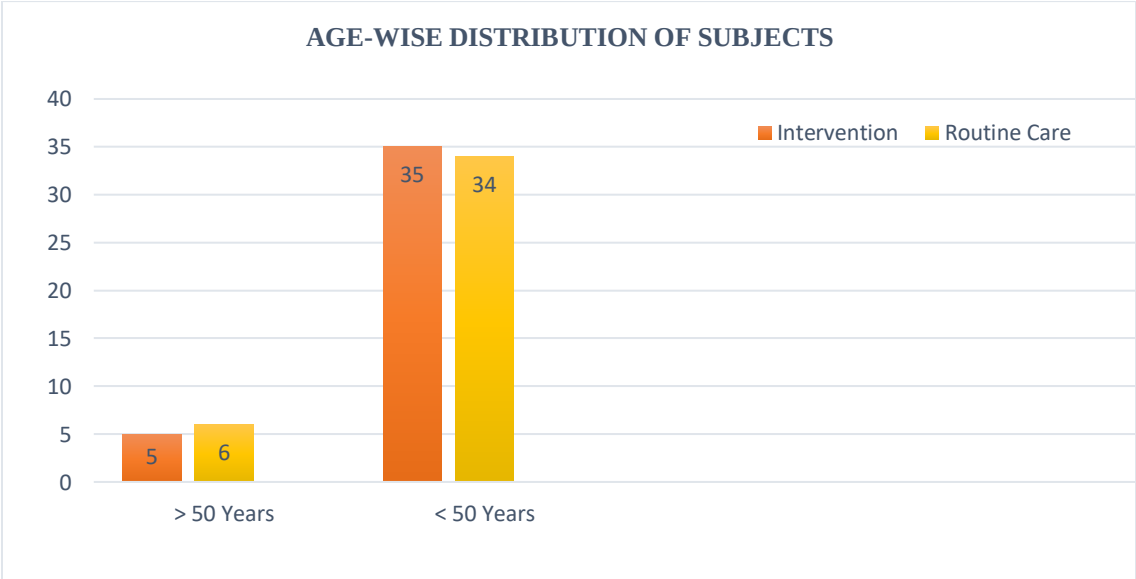

FIGURE 2 AGE-WISE DISTRIBUTION OF SUBJECTS

TABLE 3 DISTRIBUTION OF RISK FACTORS IN SUBJECTS

No. of Risk Factors	Intervention (n=40)	Routine Care (n=40)
0	3 (7.5%)	3 (7.5%)
1	7 (17.5%)	9 (22.5%)
2 & 2+	30 (75%)	28 (70%)

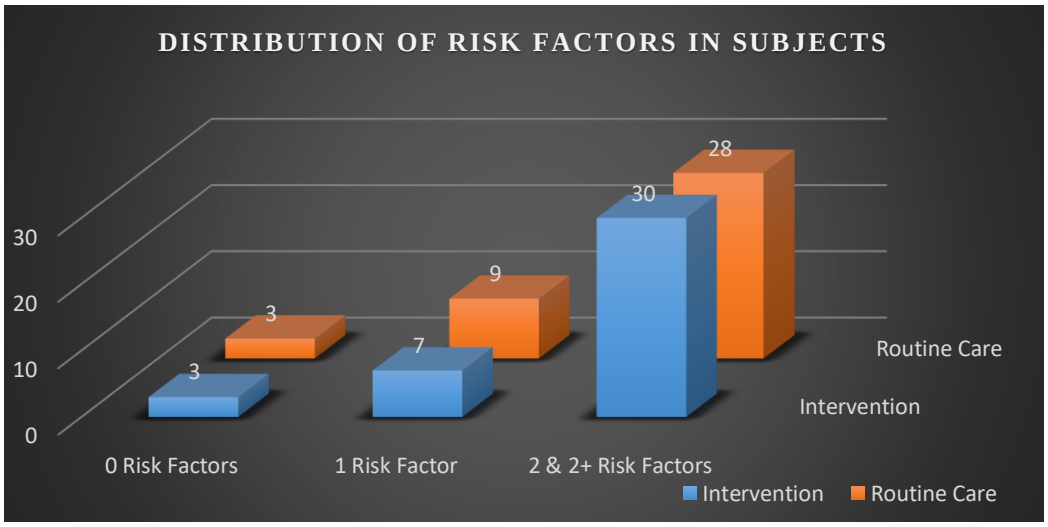

FIGURE 3 DISTRIBUTION OF RISK FACTORS IN SUBJECTS

TABLE 4 DISTRIBUTION OF RISK FACTORS CONTRIBUTED TO INCREASED LIPID LEVEL

Risk Factors	Intervention (n=40)	Routine Care (n=40)
Smoking	19	21
Diabetes Mellitus	30	34
Age (45 years or elder in Males; 55 Years or elder in Females)	24	28
Hypertension	24	27
HDL < 40 mg/dL	32	32

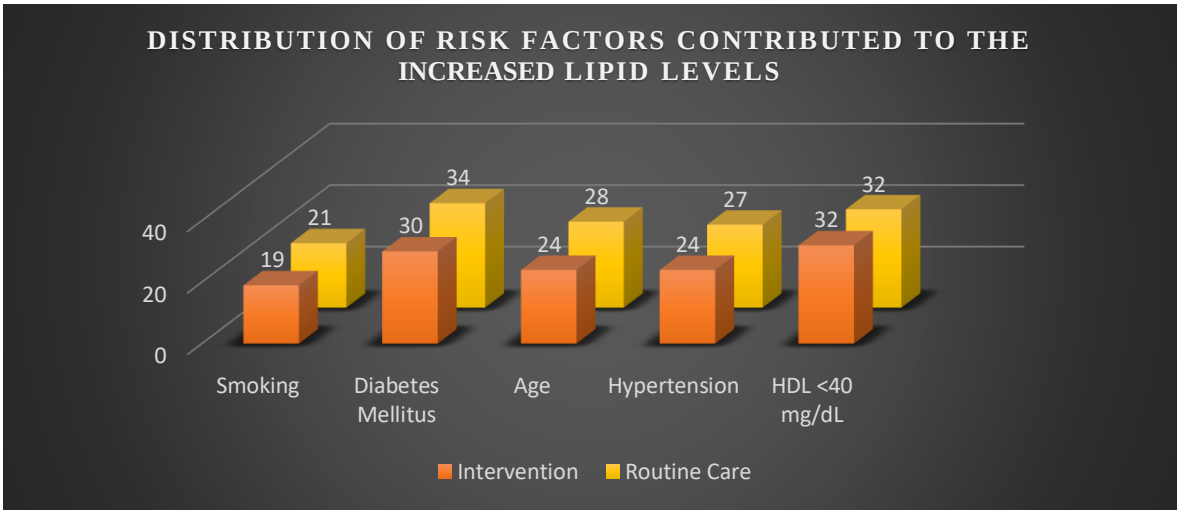

FIGURE 4 DISTRIBUTION OF RISK FACTORS CONTRIBUTED TO INCREASED LIPID LEVELS

TABLE 5 DISTRIBUTION OF LIPID LEVELS AMONG INTERVENTION GROUP

Lipid Levels (mg/dL)	Baseline (mg/dL)	Final (mg/dL)	Average Change (mg/dL)	p-value within the group
Low-Density Lipoprotein (LDL)	168 ± 32.5	89 ± 12.8	-79 ± 34.9	0.000001
High-Density Lipoprotein (HDL)	46 ± 08.5	53 ± 5.8	7 ± 10.29	0.000017
Triglycerides	184 ± 22.5	158 ± 10.5	-26 ± 24.80	0.049
Total Cholesterol	248 ± 37.5	194 ± 18.5	-54 ± 41.87	0.000001

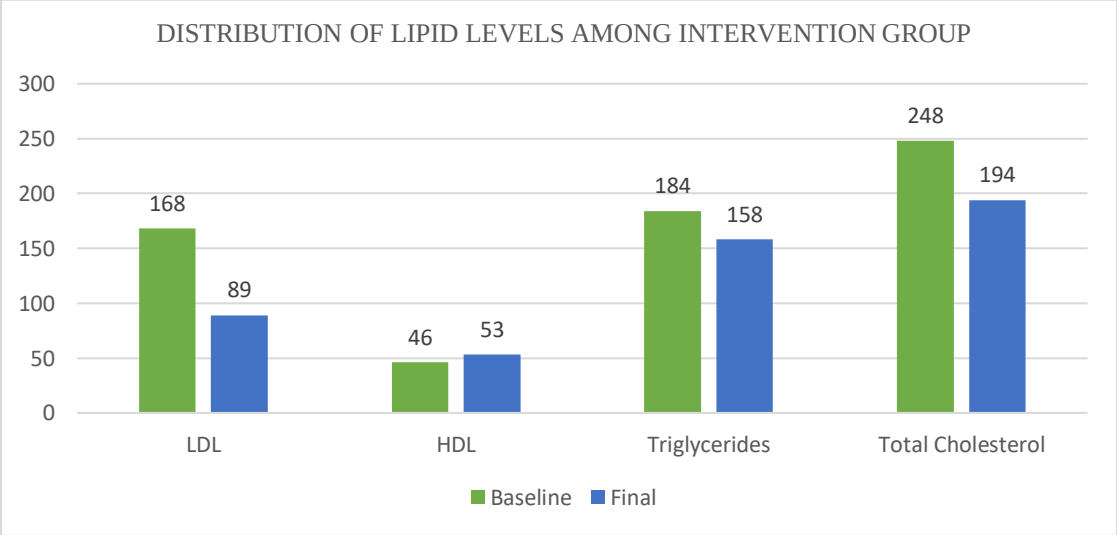

FIGURE 5 DISTRIBUTION OF LIPID LEVELS AMONG INTERVENTION GROUP

TABLE 6 DISTRIBUTION OF LIPID LEVELS AMONG ROUTINE CARE GROUP

Lipid Levels (mg/dL)	Baseline (mg/dL)	Final (mg/dL)	Average Change (mg/dL)	p-value within the group
Low-Density Lipoprotein (LDL)	172 ± 26.5	167 ± 11.8	-5 ± 29.01	0.2756
High-Density Lipoprotein (HDL)	43 ± 11.5	46 ± 3.8	3 ± 12.232	0.1172
Triglycerides	179 ± 35.5	173 ± 27.5	-6 ± 44.90	0.3981
Total Cholesterol	246 ± 30.5	239 ± 28.5	-7 ± 41.74	0.2888

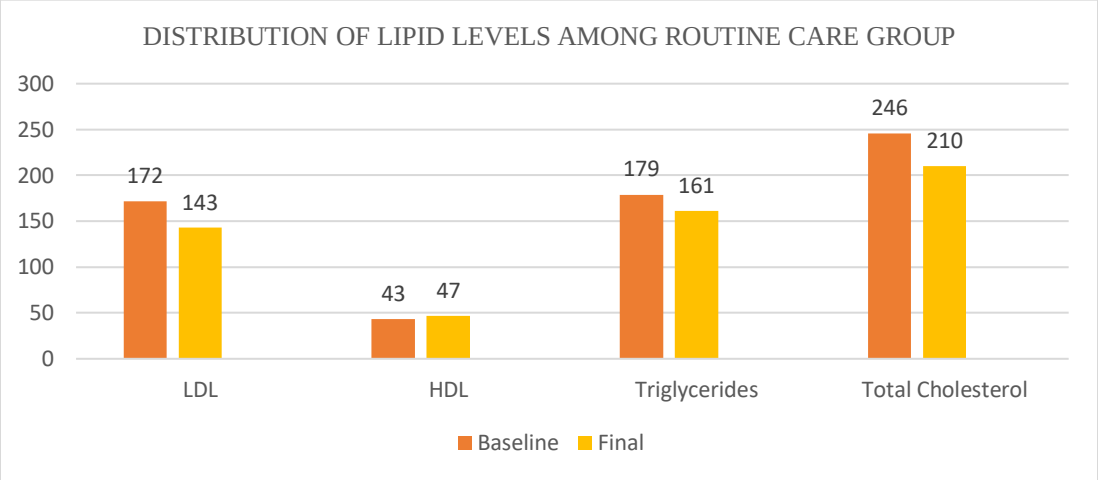

FIGURE 6 DISTRIBUTION OF LIPID LEVELS AMONG ROUTINE CARE GROUP

TABLE 7 IMPACT OF CLINICAL PHARMACIST'S COUNSELING ON LOW-DENSITY LIPOPROTEIN MANAGEMENT AMONG INTERVENTION GROUP

LDL LEVELS (mg/dL)	No. of Patients (n=40) Baseline	No. of Patients (n=40) Final
< 105	0	21 (52.5%)
105-130	9 (22.5%)	6 (15%)
131-160	15 (37.5%)	7 (17.5%)
> 160	16 (40%)	6 (15%)

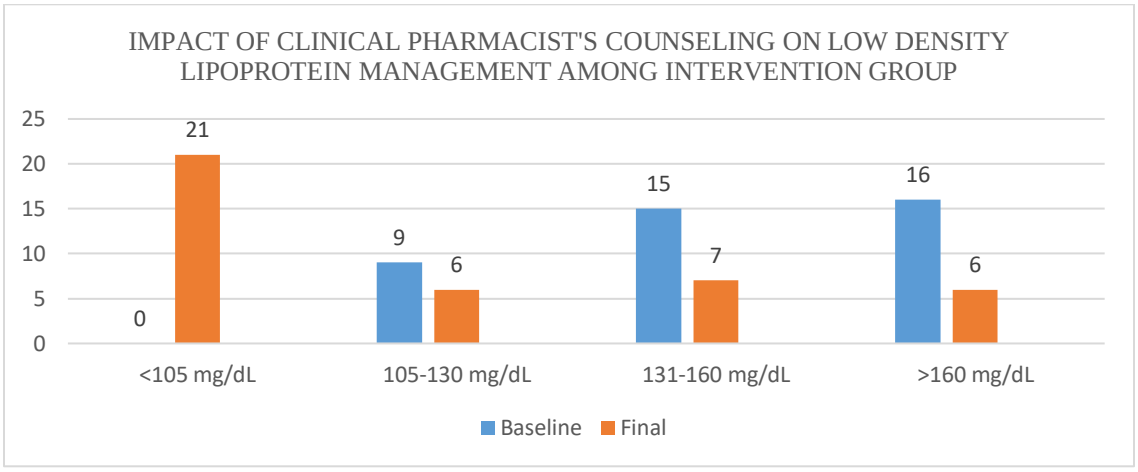

FIGURE 7 IMPACT OF CLINICAL PHARMACIST'S COUNSELING ON LOW-DENSITY LIPOPROTEIN MANAGEMENT AMONG INTERVENTION GROUP

TABLE 8 MANAGEMENT OF LOW-DENSITY LIPOPROTEIN LEVELS IN THE ROUTINE CARE GROUP WITHOUT CLINICAL PHARMACIST'S INTERVENTION

LDL LEVELS (mg/dL)	No. of Patients (n=40) Baseline	No. of Patients (n=40) Final
< 105	0	4 (10%)
105-130	13 (32.5%)	12 (30%)
131-160	12 (30%)	8 (20%)
> 160	15 (27.5%)	16 (40%)

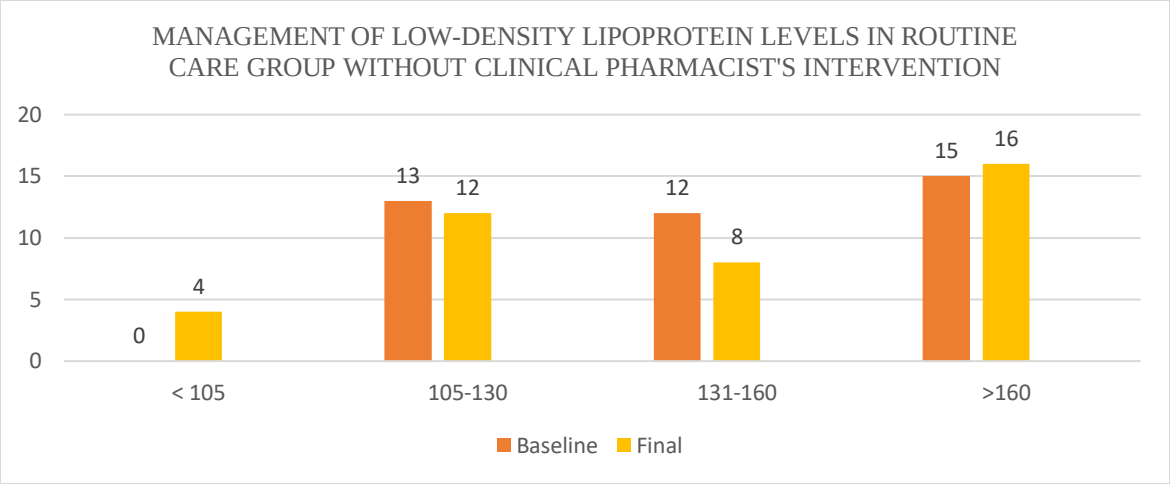

FIGURE 8 MANAGEMENT OF LOW-DENSITY LIPOPROTEIN LEVELS IN ROUTINE CARE GROUP WITHOUT CLINICAL PHARMACIST'S INTERVENTION

TABLE 9 BASELINE RESPONSES TO MORISKY MEDICATION ADHERENCE SCALE (INTERVENTION GROUP)

MMAS CATEGORY (SCORE RANGING)	No. of Subjects (n=40)	Percentage %
Low (<6)	26	65%
Medium & High (6-8)	14	35%

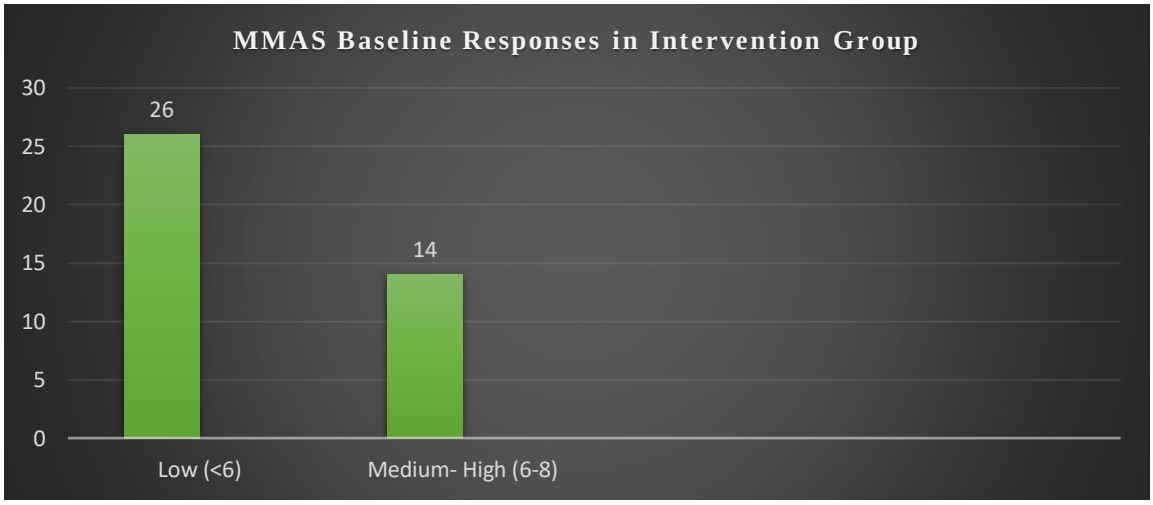

FIGURE 9 BASELINE RESPONSES TO MORISKY MEDICATION ADHERENCE SCALE (INTERVENTION GROUP)

TABLE 10 BASELINE RESPONSES TO MORISKY MEDICATION ADHERENCE SCALE (ROUTINE CARE GROUP)

MMAS CATEGORY (SCORE RANGING)	No. of Subjects (n=40)	Percentage %
Low (<6)	27	67.5%
Medium & High (6-8)	13	32.5%

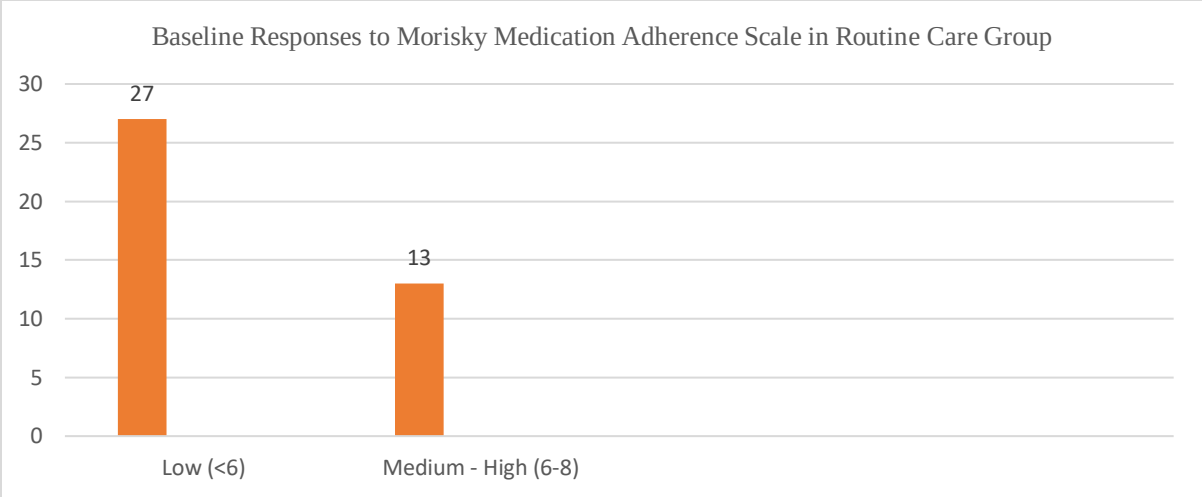

FIGURE 10 BASELINE RESPONSES TO MORISKY MEDICATION ADHERENCE SCALE (ROUTINE CARE GROUP)

TABLE 11 CHANGES IN MEDICATION ADHERENCE ACCORDING TO THE MMAS SCALE SCORING OF THE INTERVENTION GROUP AFTER 3 MONTHS OF TELEPHONIC COUNSELING

MMAS SCORE	BASELINE	FINAL (AFTER 3 MONTHS)	p-value
Total Number of Subjects with MMAS score of <6	26	3	-
MMAS Mean score (n=40)	5.15	6.85	<0.0001

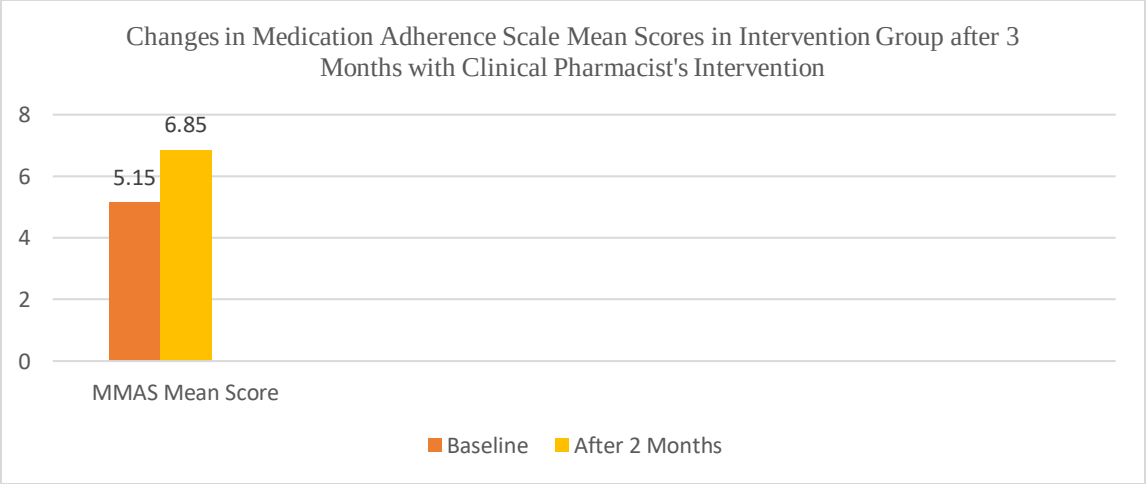

FIGURE 11 CHANGES IN MEDICATION ADHERENCE ACCORDING TO THE MMAS SCALE SCORING OF THE INTERVENTION GROUP AFTER 3 MONTHS OF TELEPHONIC COUNSELING

TABLE 12 CHANGES IN MEDICATION ADHERENCE ACCORDING TO THE MMAS SCALE; MEAN SCORES OF THE ROUTINE CARE GROUP AFTER 3 MONTHS

MMAS SCORE	BASELINE	FINAL (AFTER 3 MONTHS)	p-value
Total Number of Subjects with MMAS score of <6	27	25	-

MMAS Mean score (n=40)	5.125	5.225	0.71225

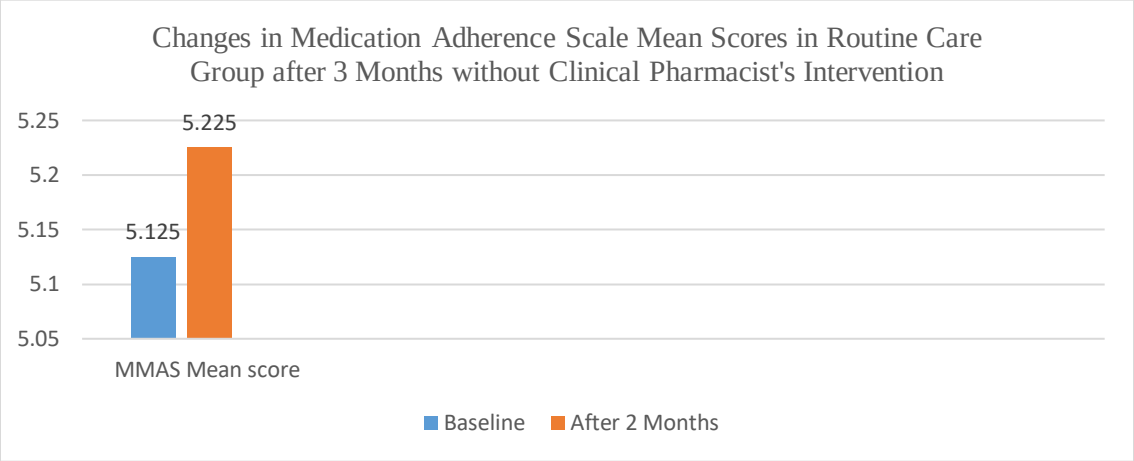

FIGURE 12 CHANGES IN MEDICATION ADHERENCE ACCORDING TO THE MMAS SCALE; MEAN SCORES OF THE ROUTINE CARE GROUP AFTER 3 MONTHS

DISCUSSION:

Controlling Hyperlipidemia is essential for avoiding overall illness consequences because it raises the risk of cardiovascular disorders. When considering both high cholesterol levels and morbidity in terms of recurrent myocardial infarction, it has been demonstrated that poor adherence and a high incidence of discontinuation are significant contributors to failed treatment. Clinical chemist interventions, among other multidisciplinary ones, are crucial for enhancing patient outcomes.

Each study group (both test and control groups) had 40 participants, and none of them were lost to follow-up. Both groups' initial characteristics were comparable. 9 female and 31 male subjects were recruited in the intervention group of 40 subjects. The control group (Routine Care Group) also included 28 male subjects and 12 female subjects, a total number of 40 subjects (Figure 1; Table 1)

Subjects in this study were divided into age groups of '50 years or under' and '50 years or older'. 40 participants made up the intervention group, of which 5 subjects were under the age of 50 years and 35 subjects were over the age of 50 years. Similar to the experimental group, the control group had 40 subjects, with 6 of them being under 50 years and 34 being over 50 years (Table 2, Figure 2).

6 subjects were randomly assigned to the intervention (3 subjects) and routine care (3 subjects) groups and none of them are affected by any risk factors. 7 subjects in the intervention group and 9 subjects in the routine care group were impacted by 1 risk factor. 30 subjects from the Intervention group and 28 subjects from the Routine Care group were affected by 2 or 2+ risk factors. Hypertension, Diabetes mellitus, age, HDL of <40mgdL, and smoking were identified to be the main risk variables that contributed to elevated lipid levels in both the intervention and routine care groups. (Table 3, Figure 3)

Smoking (19 subjects in the Intervention group and 21 subjects in the Routine Care group), Diabetes Mellitus (30 subjects in the Intervention group and 34 subjects in the Routine Care group), Age of 45 years and greater in men and 55 years and greater in women (24 subjects in Intervention group and 28 subjects in Routine Care group), Hypertension (24 subjects in Intervention group and 27 subjects in Routine Care group), and low HDL of <40 mg/dL (32 subjects in Intervention group and 32 subjects in Routine Care group) were among the risk variables that were displayed slightly more in number in the Routine care group (Control) than in the intervention group (test group). (Table 4, Figure 4).

It has been evident that Clinical Pharmacist interventions significantly improved smoking cessation rates, significantly reduced total cholesterol, significantly reduced LDL cholesterol, and significantly reduced systolic and diastolic blood pressure for major disease states and preventive health activities related to diabetes, Hyperlipidemia, and hypertension. Even while this shows the advantages of chemist intervention for a number of specific risk factors, it must be acknowledged that managing patients sometimes necessitates a simultaneous

evaluation of numerous risk variables and interventions.

The Baseline Mean readings of Low-Density Lipoprotein levels of the Intervention group were 168 ± 32.5 mg/dL. They were decreased to 89 ± 12.8 mg/dL after three months of clinical pharmacist intervention, and the average change is roughly -79 ± 34.9 mg/dL. The Baseline Mean readings of Total Cholesterol levels in the intervention group were 248 ± 37.5 mg/dL. They were lowered to 194 ± 18.5 mg/dL after three months of clinical pharmacist intervention, with an average change of -54 ± 41.87 mg/dL. At the level of significance of p-value <0.05, the reduction in Total Cholesterol and Low-Density Lipoprotein levels was found to be statistically significant.

The baseline mean readings of Triglyceride levels for the intervention group was 184 ± 22.5 mg/dL. After a clinical pharmacist's intervention for three months, the value was decreased to 158 ± 10.5 mg/dL, with an average drop of around -26 83 ± 24.82 mg/dL. Similarly to that, the baseline level of High-Density Lipoprotein was 46 ± 08.5 mg/dL. It was increased to 53 ± 5.8 mg/dL, after three months of Clinical Pharmacist intervention, with an average change of around 7 ± 10.29 mg/dL. At a p-value <0.05, it was found that changes in triglycerides and high-density lipoproteins were statistically insignificant. (Table 5)
The Baseline Total Cholesterol in the Routine Care (Control Group) was 246 ± 30.5 mg/dL. It dropped to 239 ± 28.5 mg/dL after three months, with an average reduction of around -7 ± 41.74 mg/dL. Likewise, the low-density lipoprotein baseline values were 172 ± 26.5 mg/dL. It dropped to 167 ± 11.8 mg/dL after two months; the average reduction was -5 ± 29.01 mg/dL. At a p-value of <0.05, it was determined that the decrease in total cholesterol and low-density lipoprotein levels was statistically insignificant.

The baseline triglyceride value for the routine care (Control Group) was 179 ± 35.5 mg/dL. It dropped to 173 ± 27.5 mg/dL after three months, with an average reduction of around -6 ± 44.90 mg/dL. The baseline level of high-density lipoprotein was 43 ± 11.5 mg/dL. After receiving standard therapy for three months, it was changed to 46 ± 3.8 mg/dL, with an average change of 3 ± 12.232 mg/dL. At a p-value >0.05, it was determined that changes in triglycerides and high-density lipoproteins were statistically insignificant. (Table 6)
A clinical pharmacist's management of Dyslipidemia will result in a large drop in the mean level of low-density lipoprotein. This implies that clinical pharmacist involvement in cholesterol management, including medication prescribing, Lifestyle modifications, and dietary advice leads to better clinical results, as measured by a larger reduction in Low-Density Lipoprotein levels. Clinical Pharmacists are frequently underutilized in Indian medical practices. This study demonstrates that a clinical pharmacist's active participation in the multidisciplinary medical team has a positive effect on Low-density lipoprotein lowering. This is remarkable, given the evidence that they significantly reduce the use of healthcare resources and enhance outcomes when managing chronic diseases.

There were no participants in the clinical pharmacist's intervention group who had baseline Low-Density Lipoprotein values below 105 mg/dL and were increased to 21 after three months of clinical pharmacist intervention. There were 9 subjects with baseline low-density lipoprotein values between 105 and 130 mg/dL who were reduced to 6 Subjects after 3 months of Telephonic Counseling. There were 15 participants having baseline Low-Density Lipoprotein values between 131 and 160 mg/dL and were reduced to seven after three months of the clinical pharmacist's intervention. There were 16 subjects with baseline Low-Density Lipoprotein values of more than 160 mg/dL and were reduced to 6 after 3 months of the Clinical Pharmacist's intervention (Table 7).

There were no participants in the control group who had baseline Low-Density Lipoprotein values below 105 mg/dL and were increased to 4 after three months of routine care. There were 13 subjects with baseline low-density lipoprotein values between 105 and 130 mg/dL who were reduced to 12 Subjects after 3 months of routine care. There were 12 participants having baseline Low-Density Lipoprotein values between 131 and 160 mg/dL and were reduced to 8 after three months of routine care. There were 15 subjects with baseline Low-Density Lipoprotein values of more than 160 mg/dL and were increased to 16 in number after 3 months of routine care (Table 8). This data showed that a straightforward clinical pharmacist intervention resulted in a significant improvement in the management of low-density lipoprotein and significant changes in the number of patients who were adequately treated and succeeded in attaining the goal of Low-density Lipoprotein Management.

In this study, of the 40 participants in the intervention (treatment/test) group, 26 (65%) had MMAS scores below 6, and 14 (35%) had scores between medium and high (range: 6–8). However, out of 40 subjects in the Routine care (Control) group, 27 (67.5%) had MMAS scores below 6, and 13 (32.5%) had scores

ranging from moderate to high (range: 6-8). (Table, Figures 9 and 10). But after three months of telephonic counseling by the trainee clinical pharmacists, only three subjects in the intervention (treatment/test) group had MMAS scores below six (MMAS Mean Score of 6.85). However, in the Routine care (control) group of 40 subjects, 25 subjects still remained non-adherent to the medication plan after three months of routine care (MMAS Mean Score of 5.225), up from the baseline of 27 patients (MMAS Mean Score 5.125). This data showed that a straightforward clinical pharmacist intervention significantly improved the medication adherence in the management of low-density lipoprotein cholesterol. Test group's Morisky adherence mean scores considerably rose from 5.15 to 6.85 for the Study Population of 40 subjects. The control group, however, has not experienced any substantial changes in the mean scores for the Study Population of 40 subjects (changes from 5.125 to 5.225).

CONCLUSION:

There is a critical potential for clinical pharmacists to contribute to the advancement of the productivity and viability of pharmacotherapy in patients with Dyslipidemia. As illustrated in this study, intriguing medical groups that incorporate clinical pharmacists in lipid management achieved more prominent decreases in LDL for patients who have been evaluated with multiple risk factors compared to patients without clinical pharmacists' Management of Dyslipidemia. Additionally, clinical pharmacist-delivered intervention progressed in the understanding of Medication adherence and improved patient compliance. Dynamic Active support by clinical pharmacists in lipid management for all patients with elevated LDL comes about resulting in improved intermediate outcomes in achieving lipid targets. These results may result eventually in diminished long-term cardiovascular events and enhanced quality of life for patients with Dyslipidemia as well as reduced long-term costs related to sequelae of Dyslipidemia. Expanded treatment productivity within the administration of Dyslipidemia by clinical pharmacists may allow other healthcare providers to address and oversee other perspectives of their patient's well-being.

Conflict of Interest:

the authors declare no conflict of interest

REFERENCES:

1. The Lipid Research Clinics Coronary Primary Prevention Trial results I. Reduction in Incidence of coronary heart disease. *Journal of American Medical Association*. 1984;251: 351-364
2. The Lipid Research Clinics Coronary Primary Prevention Trial results II. The relationship of reduction in Incidence of coronary heart disease to Cholesterol Lowering. *Journal of American Medical Association.* 1984; 251:365-374
3. Frick M, Elo O, Haapa K, Heinonen O, Heinsalmi P, Helo P, Helsinki Heart Study: Primary Prevention Trial with Gemfibrozil in Middle-aged Men with Dyslipidemia. Safety of Treatment, changes in risk factors and incidence of Coronary Heart disease. Journal of American Medical Association. 1987; 317:1237-45
4. Pedersen T, Kjekshus J, Berg K. Randomized trial of cholesterol lowering in 4,444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). Lancet. 1994;344(8934):1383-9.
5. Sacks F, Pfeffer M, Noye L and the CaRECT Investigators. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. New England Journal of Medicine. 1996;335:1001-9.
6. Shepherd J, Cobbe S, Ford 1, and the West of Scotland Coronary Prevention Study (WOSCOPS) Group. Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia.New England Journal of Medicine. 1995;333:1301-7.
7. Downs J, Clearfield M, Weiss S, et al. Primary prevention of acute coronary events with lovastatin in men and women with average cholesterol levels: results of AFCAPS/TesCAPS. Journal of American Medical Association.1998;279:1615-22.
8. Superko R. Prevention and regression of atherosclerosis with drug therapy. Clinical Cardiology. 1991;14:40-7.
9. Superko H, Krauss R. Coronary artery disease regression: convincing evidence for the benefit of aggressive lipoprotein management. Circulation.1994:90:1056-693.
10. Andrade SE, Walker AM, Gottlieb LK, HollenbergNK, Testa MA, Saperia GM, et al. Discontinuation of antihyperlipidemic drugs: do rates reported in clinical trials reflect rates in primary care settings?New England Journal of Medicine.1995;332:1125-1131.
11. Machado M, Nassor N, Bajcar JM, Guzzo GC, Einarson TR. Sensitivity of patient outcomes to pharmacist interventions. Part III: systematic review and meta-analysis in hyperlipidemia management. The Annals of Pharmacotherapy.2008; 42(9):1195-1207.
12. Padiyara RS, DSouza JJ, Rihani RS.Clinical pharmacist intervention and the proportion of

diabetes patients attaining prevention objectives in a multispecialty medical group. Journal of Managed Care Speciality Pharmacy.2011; 17(6): 456-462.
13. Keefe JH, Cordain L. Cardiovascular disease resulting from a diet and lifestyle at odds with our Paleolithic genome: how to become a 21st-century hunter-gatherer.Mayo Clinical Proceedings.2004, 79: 101-108.
14. Cordain L, Eaton SB, Miller JB, et al. The paradoxical nature of hunter-gatherer diets: meat-based, yet non-atherogenic.European Journal of Clinical Nutrition.2002; 56(1): S42-S52.
15. O'Keefe JI, Cordain L, Harris WH, et al. Optimal low-density lipoprotein is 50 to 70 mg/dl: lower is better and physiologically normal. Journal of American College Cardiology.2004; 43: 2142-2146.
16. Brown L, Rosner B, Willett WW, et al. Cholesterol-lowering effects of dietary fiber: a meta-analysis.American Journal of Clinical Nutrition.1999; 69:30-42.
17. Demonty I, Ras RT, van der Knaap HC, et al. Continuous dose-response relationship of the LDL-cholesterol-lowering effect of phytosterol intakeJournal of Nutrition.2009;139: 271-284.
18. Sabate J, Oda K, Ros E. Nut consumption and blood lipid levels: a pooled analysis of 25 intervention trials. Archives of Internal Medicine.2010;170: 821-827.
19. Taku K, Umegaki K, Sato Y, et al. Soy isoflavones lower serum total and LDL cholesterol in humans: a meta-analysis of 11 randomized controlled trials. American Journal of Clinical Nutrition.2007, 85: 1148-1156.
20. Williams PT, Krauss RM, Kindel-RK2227)S, et al. Relationship of dietary fat, protein, cholesterol, and fiber intake to atherogenic lipoproteins in men. American Journal of Clinical Nutrition.1986; 44: 788-797.
21. Siri PW, Krauss RM. Influence of dietary carbohydrate and fat on LDL and HDL particle distributions. Current Atherosclerosis Reports.2005; 7:455-459.
22. Mozaffarian D, Aro A, Willett WC. Health effects of trans-fatty acids: experimental and observational evidence.European Journal of Clinical Nutrition.2009; 63(2): $5-S21.
23. Richard C, Couture P, Desroches S, et al. Effect of the Mediterranean diet with and without weight loss on surrogate markers of cholesterol homeostasis in men with the metabolic syndrome. British Journal of Nutrition.2012; 107:705—711.
24. Georgousopoulou EN, Pitsavos C, Panagiotakos D, et al. Adherence to Mediterranean is the most important protector against the development of fatal and non-fatal cardiovascular event: 10-year follow-up (2002- 2012) of the Attica study. Journal of the American College of Cardiology.2015; 65: 1449.
25. Ornish D, Brown SE, Scherwitz LW, et al. Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. Lancet.1990; 336: 129-133.
26. Belardinelli R, Paolini 1, Cianci G, et al. Exercise training intervention after coronary angioplasty: the ETICA trial. Journal of the American College of Cardiology.2001; 37: 1891-1900.
27. Wosornu D, Bedford D, Ballantyne D. A comparison of the effects of strength and aerobic exercise training on exercise capacity and lipids after coronary artery bypass surgery. European Heart Journal.1996; 17: 854-863.
28. Yu CM, Li LS, Ho HH, et al. Long-term changes in exercise capacity, quality of life, body anthropometry, and lipid profiles after a cardiac rehabilitation program in obese patients with coronary heart disease. American Journal of Cardiology.2003; 91:321-325
29. Wood PD, Stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise.New England Journal of Medicine.1988; 319: 1173-1179.
30. Halle M, Berg A, Konig D, et al. Differences in the concentration and composition of low-density lipoprotein subfraction particles between sedentary and trained hypercholesterolemic men. Metaholism.1997; 46:186—-I91.
31. Williams PT, Krauss RM, Vranizan KM, et al. Effects of exercise-induced weight loss on low density lipoprotein subfractions in healthy men. Arteriosclerosis.1989;9: 623-632.
32. Kraus WE, Houmard JA, Duscha BD, et al. Effects of the amount and intensity of exercise on plasma lipoproteins.New England Journal of Medicine.2002; 347: 1483-1492.
33. Ahmed HM, Blaha MJ, Nasir K, et al. Effects of physical activity on cardiovascular disease.American Journal of Cardiology.2012; 109: 288-295.
34. Scirica BM, Cannon CP. Treatment of elevated cholesterol.Circulation.2005;111:360-363.