World Journal of Current Medical and Pharmaceutical Research
Content available at www.wjcmpr.com ISSN: 2582-0222

A STUDY OF CARDIOVASCULAR RISK IN DYSLIPIDEMIA PATIENTS AND ROLE OF STATINS IN LOWERING THE LIPID LEVELS.

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Article History

Received on: 22-12-2024
Revised on: 04-01-2025
Accepted on: 12-02-2025

Abstract

This study aimed to predict CVD risk in dyslipidemia patients based on factors like HTN, DM, hypothyroidism, alcohol, smoking, and obesity. We recruited 75 dyslipidemia patients (July–December 2023) and analyzed their CVD risk via 2D-ECHO & ECG. Statin combination therapy (Ecosprin, Ecosprin + Rosuvastatin) was prescribed for CVD-confirmed patients, while statin monotherapy (Atorvastatin) was given to those without CVD risk, aligning with Kirsten Bibbins-Domingo et al.'s study. Participants were categorized by age: 18–30 (11), 31–43 (17), 44–56 (22), and >56 (25). Gender-wise, females (40) outnumbered males (35), similar to Seyed Mahmoud Latifi et al.'s study. Based on residence, 47.67% were urban and 57.53% rural, with rural participants being higher. HTN was found in 52 patients, with 34 at CVD risk. DM was present in 34, with 24 at risk, aligning with Razieh Anari et al.'s findings. Hypothyroidism affected 22 patients, with 11 at risk, similar to Fatima Tarboush et al.'s study. Obesity was noted in 24 patients, with 17 at risk. Alcohol consumption was reported in 36 patients, with 28 at risk, while 31 were smokers, with 23 at risk. These findings highlight significant CVD risk factors in dyslipidemia patients.

Keywords: Dyslipidemia, Statin Combination Therapy, Kirsten Bibbins-Domingo, Hypothyroidism

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DOI: https://doi.org/10.37022/wjcmpr.v7i1.355

Introduction

Dyslipidemia, which comprises high plasma triglyceride levels, low HDL cholesterol concentrations, and increased tiny dense LDL cholesterol particles, contributes significantly to the development of CHD.

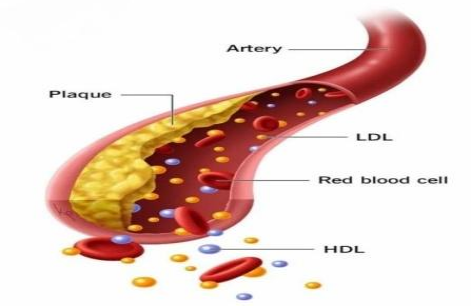

Fig: 01 Dyslipidemia

Types of Dyslipidemia

Primary dyslipidaemia is a condition where up to 60% of the discrepancy in cholesterolfasting lipids is believed to have a genetic basis, with its expression which includes interactions due to external elements. Typical hereditary illness in this category include primary hypercholesterolemia's, characterized by elevated LDL-C, Primary mixed hyperlipidemia's', involving raised levels of LDL-C and TG and primary hypertriglyceridemia's, including Type 3 hyperlipoproteinemia, both hereditary apoC-II deficiency and familial lipoprotein lipase insufficiency.

Familial Hypercholesterolemia

Heterozygous FH is an inherited metabolic disorder, estimated to affect around 1 in 500 of the population, although recent data suggests a higher prevalence. This condition results from various mutations in genes responsible for clearing LDL-C, with the LDL receptor gene being the most commonly affected. LDL-C of FH individuals ranges more than twice or thrice of the normal people. Early identification and treatment are crucial, as individuals with FH face elevated LDL-C concentrations and associated risks. It is inherited dominantly, with a 50% risk for siblings and children of affected parent. Diagnosis relies on

clinical criteria, and specialist advice is recommended for high-risk patients.

In heterozygous FH, (20 years) quick manifestation of cardiovascular disease is observed when compared to the healthy population, often leading to atherosclerotic heart disease, particularly in men. Symptoms like corneal arcus, tendon xanthoma, and xanthelasma typically appear in thethird decade. On the other hand, homozygous FH is uncommon and linked to a near total incapacity to eliminate LDL-C, which causes early-onset aortic abnormality, cutaneous and tendon xanthomas, and a high risk of myocardial infarction.

Familial Combined Hyperlipidaemia

It involves 1 in 50–200 people with excessivesynthesis of VLDL-C, leading to elevated levels of triglycerides, LDL-C, apoB & small dense LDL particles. It increases events of atherosclerosis and is observed among 10% subjects of coronary heart disease in early 60's.

Familial Type III Hyperlipoproteinemia

In these individuals 1 in 5000–10,000 are expressed with aggregation of chylomicron and VLDL residue due to less active forms of apoE. Elevated triglycerides and TC, along with physical manifestations like corneal arcus, xanthelasma, tuberoeruptive xanthomas, and palmar striae, predispose individuals with early onset of atherosclerosis.

Familial Lipoprotein Lipase Deficiency

In every 1 per 1 million individuals, presents inchildhood with marked hypertriglyceridemia and chylomicronemia. It results from the insufficiency of lipoprotein lipase, leading to recurrent abdominal pain, eruptive xanthomas, lipemia retinalis, and an enlarged spleen. Acute pancreatitis is the major complication, but there is no increased susceptibility to atherosclerosis.

Secondary Dyslipidemia

Up to 40% of dyslipidemias result from secondary causes such as various disorders, dietary choices, or drug therapy side effects. Fortunately, correcting the underlying conditions, implementing efficacious nutritional suggestion , or withdrawing the undesirable medication to often rectify lipid abnormalities in secondary dyslipidemia. In some cases, a disorder maybe associated with dyslipidemia without causing it, as seen in the coexistence of hyperuricemia (gout) and hypertriglyceridemia.

Epidemiology

Hypercholesterolemia, the most prevalent type of Dyslipidemia, is linked to heightened chances of CVD. Raised LDL-C, identified as the 15th most emerging cause of death in 1999 , climbed to the 11th position in 2007 and further rose tothe 8th spot in 2019 . WHO stated that 39% of adults worldwide have elevated blood cholesterol, with a slightly higher prevalence among women (40%) than men (37%). Elevated low-density lipoprotein (LDL) cholesterol contributed to 7.7% of global deaths in 2017, totaling 4.32 million fatalities.

Fig:02 Global Percentage Prevalance of dyslipidemia

In Saudi Arabia, the 2019 World Health Survey revealed varying cholesterol prevalence, ranging from 42% in urban to 47% in rural areas. The survey also noted that individuals with higher education and wealth exhibited lower cholesterol levels. The prevalence of dyslipidemia has raised over the last three decades worldwide. [10]

Statins

Statins stand as the fundamental approach for lowering lipid levels, has numerous extensive , randomised , placebo control trails have consistently showcased their potent effectiveness in preventing cardiovascular disease events [3].

Apart from a dietary management and healthy lifestyle, pharmaceutical approaches also helps in reduction of LDL-C. These strategies encompass medications like statins , fibrates , ezetimibe , cholestyramine and torcetrapib . Among these , statins are the most widely used , primarily works by deactivation of HMG CoA reductase to lower cholesterol synthesis. Notably statins also exhibit ant inflammatory effects with in atherosclerotic plaques, decrease acute phaseprotein levels in the blood stream and contribute with reduced probability of CVD related mortality and morbidity. Usually advised statins include atorvastatin, simvastatin and rosuvastatin. [4]

Atorvastatin

HMG-CoA reductase inhibitors, called as statins, play a pivotal role in primary, secondary, and tertiary prevention of coronary heart disease. Atorvastatin competes with HMG-CoA reductase, reducing cholesterol production through inhibiting the transformation of HMG-CoA to mevalonatein the liver. Additionally, atorvastatin enhances the number of LDL receptors on hepatocytes.

The present study was on to identify the risk of cardiovascular disease in dyslipidemia patients and to evaluate the role of statins in lowering the lipid levels.

1. To predict the risk of CVD in dyslipidemia patients.
2. To bring awareness in patients to overcome CVD
3. To study the lipid profile before and after the statins therapy.

Methodology

Study Design

Prospective observational study.

Study Site

This study is being carried out in both In-Patient and Out-Patient facilities of Department of Trident Hospital in Perecherla, Guntur.

Study Period and Duration

Our study is being carried out for a period of 6 months during the months

Study Population

71Patients visiting the In-Patient and Out-Patient facilities of Department of General medicine, diagnosed with dyslipidemia recruited based on the specific selection criteria.

Selection of Subjects

Subjects will be selected for the study based on the following inclusion and exclusion criteria

Inclusion Criteria

- Patients who are diagnosed with dyslipidemia.
- Adults of age above 18 diagnosed with dyslipidemia are included.
- Diabetes mellitus, Hypertension and Hypothyroidism patients diagnosed with dyslipidemia.
- Patients who does not have any history of CVD.

Exclusion Criteria

- Patients with different comorbidities other than HTN, DM& Hypothyroidism.
- Patients having known history of CVD.
- Patients under the age 18 are excluded.

Materials

We have conducted a prospective observational study in the In-Patient and Out-Patient facilities of Department of General medicine and Cardiology, Trident Hospital in Perecherla, Guntur.. This study was conducted for a period of six months during the months of with the approval from institutional human ethics committee (IHEC). We recruited 75 patients under the physician's guidance based on the inclusion and exclusion criteria.

Following materials are used in this study

- Patient consent from
- Data collection form
- Patient information leaflet

Patient consent form: Patient consent forms, which include study details, were created in four languages: Telugu, Hindi, Urdu, and English, for the convenience of the patients.

Patients were enlisted by signing the consent form after being granted permission to take part in the research study, which contained information about the study.

Data collection form: An institutional professor and a hospital physician were consulted in the preparation of the data collection form. Within a small study population group, the designed data collection form was assessed to ensure it was appropriate for the current investigation. The following patient information, which is crucial, was gathered in relation to the specially created data collection form.

Demographic details: include name, age, gender, date of visit, OP number and residence address of the patient for patient identification and socioeconomic background.

History of present illness to acquire duration of complaints specific to the patient.

Past medical and medication history to know former health related issues of the patient and whether the patient was administered or administering any medication.

Family history to know whether any of family members of the patient had history of any disease condition.

Signs and symptoms to know the symptoms regarding the disease condition of the patient.

General Examination and vitals to assess and monitor patient condition at the time of consult.

Lipid profile to evaluate the lipid levels (TC, LDL ,HDL,TG, VLDL) ECG&2DECHO to find the abnormalities present in heart funtions. Patient information leaflet

Patient information pamphlets, providing a brief information about disease and its risk factors, signs and symptoms, diet and lifestyle changes and information about statins and these were distributed to patients during counseling sessions.

Results

There were 7 dropouts and the final study population was of 75 patients

1. Distribution of Study Population Based on Age Groups

Gender	Subjects	%Subjects
Male	35	46.7%
Female	40	53.3%

2: Distribution of Study Population Based on Gender.

Age Group	18-30	31-43	44-56	56 above
Subjects	11	17	22	25

3: Distribution of Study Population Based on Residence.

Residence	No of Subjects	%subjects
Urban	32	42.67%
Rural	43	57.33%

4. Distribution of Study Population Based on Signs & Symptoms

5: Distribution of Study Population Based on DM

signs & symptoms	No of subjects	% subjects
vomiting, Nausea	6	2.35%
Chest pain	12	4.71%
shortness of breath	18	7.06%
dizziness, heart palpitations	19	7.45%
pain, tightness, pressure in the neck, jaw, & shoulders	20	7.84%
tightness or pressure in the chest	27	10.59%
swelling in legs, ankles, feet, stomach & veins of the neck	28	10.98%
symptoms may get worse with activity or stress	29	11.37%
sleep problems & daytime exhaust	43	16.86%
indigestion & Heartburn	53	20.78%

6: Distribution of Study Population Based on

Parameter	No of Positives	% Positive	No of Negatives	% Negative
DM	34	45.33%	41	54.66%

Hypothyroidism

Parameter	No of Positives	% Positive	No of Negatives	% Negative
Hypothyroidism	22	29.33%	53	70.66%

7. Distribution of Study Population Based on HTN

Parameter	No of Positives	% Positive	No of Negatives	% Negative
HTN	52	73.33%	23	30.66%

8: Distribution of Study Population Based on Obesity

Parameter	No of Positives	% Positive	No of Negatives	% Negative
Obesity	24	32%	51	68%

9: Distribution of Study Population Based on Alcohol

Parameter	No of Positives	% Positive	No of Negatives	% Negative
Alcohol	36	48%	39	52%

10: Distribution of Study Population Based on Smoking

Parameter	No of Positives	% Positive	No of Negatives	% Negative
Smoking	31	41.33%	44	58.66%

11: Distribution of Study Population Based on their lipid profiles before and after Statins treatment.

Subjects	lipid profile				
	TC	HDL	TG	LDL	Vldl
avg (before)	251.12	52.7	283.2	164.3	45.24
avg(after)	223.34	49.68	261.08	137.32	43.35

12: Distribution of Study Population Based on CVD

Parameter	# Positive	% Positive	# Negative	% Negative
CVD	41	54.66%	34	45.33%

13: Distribution of Study Population Based on Factors considered in dyslipidemia patients to verify CVD

Parameters	Positive	Negative
HTN	52	23
DM	34	41
Hypothyroidism	22	53
Obesity	24	51
Alcohol	36	39
Smoking	31	44
CVD	41	34

14.Distribution of Study Population Based on the Distribution of Subjects resulted in CVD risk based on the Factors considered

Parameters	Subjects
DM	34
DM with CVD	24
HTN	52
HTN with CVD	34
Hypothyroidism	22
HY with CVD	11
Obesity	24
obesity with CVD	17
Alcohol	36
Alcohol with CVD	28
Smoking	31
Smoking with CVD	23

Discussion

In this study, the objectives were to predict the risk of CVD in dyslipidemia patients based on the factors considered like HTN, DM, Hypothyroidism, Alcohol, Smoking, and Obesity to prevent the patients from CVD

We recruited75 subjects of dyslipidemia patients for our study between july 2023 to december2023 and analysed for CVD risk based on 2DECHO&ECG, Statin combination therapy (Ecosprin,Ecosprin+Rosuvastatin)was prescribed in case of patients conformed with CVD and Statin mono therapy(Atorvastatin) in case of dyslipidemia patients without CVD risk, the study which is similar to the study conducted by Kirsten Bibbins- Domingo, PhD, MD, MAS et al

Socio-Demographic Characters of the Study Population Based On Age

In this study, we have considered the participants of age above 18 and we categorized the age into four groups i.e 18-30(11) , 31-43(17), 44-56(22) & above 56(25).

Socio-Demographic Characters of the Study Population Based On Gender

In this study , we have considered the participants of both male(35) and female(40),females were found to be more in number, which is similar to the study performed by Seyed Mahmoud Latifi, Leila Moradi, Hajieh Shahbazian, Armaghan Moravej Aleali.

Socio-Demographic Characters of the Study Population Based On Residence

In this study, we have considered the participants based on their residence, Urban (47.67%) and rural (57.53%), urban participants were found to be lower than rural participants.

Socio-Demographic Characters of The Study Population Based On HTN

Out of 75 patients 52 were found to be hypertensive and among those 34 patients were at CVD risk. Which is similar to the study conducted by Razieh Anari a, Reza Amani b, Seyed Mahmoud Latifi c, Masoud Veissi d, Hajieh Shahbazian c

Socio-Demographic Characters of the Study Population Based on DM

Out of 75 patients 34 were found to be diabetic and among those 24 patients were at CVD risk. . Which is similar to the study conducted by Razieh Anari a, Reza Amani b, Seyed Mahmoud Latifi c, Masoud Veissi d, Hajieh Shahbazian c

Socio-Demographic Characters of the Study Population Based On Hypothyroidism

Among the Study population of 75 patients 22 were found to be with Hypothyroidism and from those 11 were at CVD risk. Which is similar to the study of Fatima Tarboush, MDa, Mohammad Alsultan, MDb,*, Zaynab Alourfi, PhDc

Socio-Demographic Characters of the Study Population Based On Obesity

In 75 patients of study population 24 were obese and 17 from those were at CVD risk. Which is similar to the study conducted by Razieh Anari a, Reza Amani b, Seyed Mahmoud Latifi c, Masoud Veissi d, Hajieh Shahbazian c

Socio-Demographic Characters of the Study Population Based On Alcohol.

36 patients were found to be alcoholic among 75 patients of study population and among those 28 were at CVD risk.

Socio-Demographic Characters of the Study Population Based On Smoking.

Out of 75 patients of study population 31 has the habit of smoking and among those 23 were at risk of CVD.

Conclusion

In this study ,we have analyzed CVD risk by using tests like 2D ECHO Screening and ECG in dyslipidemia patients(who have conformed through lipid profile test).We have found that dyslipidemia patients along with underlying risk factors (HTN, DM, Hypothyroidism, Obesity, Alcohol ,Smoking) have high prevalence of CVD. Dyslipidemia patients with and without CVD were treated with Statin combination therapy and Statin mono therapy respectively and we have concluded that, after initiation of statin treatment the elevated lipid levels were reduced. We aim to enhance understanding of dyslipidemia management guidelines to prevent future CVD issues.

Acknowledgement

We sincerely thank to O.SHAKUNTHALA DEVI. Secretary and Correspondent, for her invaluable support and guidance throughout work. Her encouragement and assistant have been instrumental in our progress.

References

1. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with MI in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364:937–52.
2. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final report. Circulation. 2002:106–421.
3. Grundy SM, Cleeman JI, Merz CN, Brewer HB, Jr, Clark LT, Hunninghake DB, et al. National Heart, Lung, and Blood Institute; American College of Cardiology Foundation; American Heart Association. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. Circulation. 2004;110:227–39.
4. Brunzell JD, Davidson M, Furberg CD, Goldberg RB, Howard BV, Stein JH, et al. American Diabetes Association; American College of Cardiology Foundation. Lipoprotein management in patients with cardiometabolic risk: consensus statement from the American Diabetes Association and the American College of Cardiology Foundation. Diabetes Care. 2008;31:811–22.
5. Baigent C, Keech A, Kearney PM, Blackwell L, Buck G, Pollicino C, et al. Cholesterol Treatment Trialists' (CTT) Collaborators. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90 056 participants in 14 randomised trials of statins. Lancet. 2005;366:1267–78.
6. LaRosa JC, Grundy SM, Waters DD, Shear C, Barter P, Fruchart JC, et al. Treating to New Targets (TNT) Investigators. Intensive lipid lowering with atorvastatin in patients with stable coronary disease. N Engl J Med. 2005;352:1425–35.
7. Cannon CP, Braunwald E, McCabe CH, Rader DJ, Rouleau JL, Belder R, et al. Pravastatin or Atorvastatin Evaluation and Infection Therapy-Thrombolysis in Myocardial Infarction 22 Investigators. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. N Engl J Med. 2004;350:1495–504.
8. Gordon DJ, Probstfield JL, Garrison RJ, Neaton JD, Castelli WP, Knoke JD, et al. High-density lipoprotein cholesterol and cardiovascular disease. Four prospective American studies. Circulation. 1989;79:8–15.
9. Sarwar N, Danesh J, Eiriksdottir G, Sigurdsson G, Wareham N, Bingham S, et al. Triglycerides and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies. Circulation. 2007;115:450–8.
10. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. JAMA. 2007;298:309–16.
11. Briel M, Ferreira-Gonzalez I, You JJ, Karanicolas PJ, Akl EA, Wu P, et al. Association between change in high density lipoprotein cholesterol and cardiovascular disease morbidity and mortality: systematic review and meta-regression analysis. Br Med J. 2009;338:b92.
12. Cohen JD, Cziraky MJ, Jacobson TA, Wallace A, Cai C. Changes in the prevalence of abnormal lipid fractions among US adults: results from the National Health and Nutrition Examination Survey II, III and 1999–2006. Circulation. 2008;118:S_1081–2.
13. Centers for Disease Control and Prevention (CDC) National Diabetes Surveillance System. [Accessed June 2007];Diabetes data and trends.
14. Miller M. Disorders of hypertriglyceridemia. In: Kwiterovich PO, editor. The Johns Hopkins Textbook of Dyslipidemia. Baltimore, MD, Lippincott, Williams & Wilkins: 2009.
15. Lamarche B, Tchernof A, Mauriège P, Cantin B, Dagenais GR, Lupien PJ, et al. Fasting insulin and apoplipoprotein B levels and low-density lipoprotein particle size as risk

factors for ischemic heart disease. JAMA. 1998;279:1955–61.

16. Sattar N, Williams K, Sniderman AD, D'Agostino R, Jr, Haffner SM. Comparison of the associations of apolipoprotein B and non-high-density lipoprotein cholesterol with other cardiovascular risk factors in patients with metabolic syndrome. Circulation. 2004;110:2687–93.
17. Sierra-Johnson J, Somers VK, Kuniyoshi FH, Garza CA, Isley WL, Gami AS, et al. Comparison of apoliprotein B-apolipoprotein-AI in subjects with versus without the metabolic syndrome. Am J Cardiol. 2006;98:1369–73.
18. Miller M, Cannon CP, Murphy SA, Qin J, Ray KK, Braunwald E. PROVE-IT TIMI 22 Investigators. Impact of triglyceride levels beyond low-density lipoprotein cholesterol after acute coronary syndrome in the PROVE-IT TIMI 22 trial. J Am Coll Cardiol. 2008;51:724–30.
19. Jacobson TA, Case CC, Roberts S, Buckley A, Murtaugh KM, Sung JC, et al. Characteristics of US adults with the metabolic syndrome and therapeutic implications. Diabetes Obes Metab. 2004;6:353–62.
20. Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA, et al. American Heart Association Nutrition Committee. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. Circulation. 2006;114:82–96.