pISSN 2320-6071 | eISSN 2320-6012

Original Research Article

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20244110

Unravelling the risk factors and treatment patterns of dyslipidemia in India (UNICORN study)

Mayur Mayabhate*, Akhilesh Sharma

Medical Affairs, Alkem Labs Mumbai, Maharashtra, India

Received: 14 October 2024 Revised: 04 December 2024 Accepted: 09 December 2024

*Correspondence:

Dr. Mayur Mayabhate,

E-mail: mayur.mayabhate@alkem.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: The UNICORN study aims to determine the risk factors and patient demographics for dyslipidemia and assess the efficacy of the prevalent therapies in managing dyslipidemia.

Methods: The UNICORN study employed a retrospective cross-sectional design, analyzing data from 9,998 patient health records retrieved from tertiary care centers across India. Demographic variables, lipid profile parameters, risk factors (smoking status, alcohol consumption and dietary habits) and healthcare-related variables (treatment history of medication, lifestyle interventions and lipid profiles) were extracted from medical records. Descriptive statistics and hypothesis testing were used to assess lipid profiles, comorbidities and treatment impact. Odds ratios and paired t-tests evaluated morbidity risk and treatment efficacy.

Results: Statins were prescribed to 99.19% of the patients. 61.61% of the population did not follow any particular diet. Lipid parameters were better in obese and overweight patients, compared to normal and underweight patients (p<0.001). Patients with hypertension (239.5 vs 223.4 mg/dl), coronary artery disease (240.5 vs 231.4 mg/dl), stroke (230.3 vs 236.3 mg/dl) and peripheral arterial disease (238.1 vs 234.1 mg/dl) showed significantly higher total cholesterol levels. The odds ratio (OR) reported a higher likelihood of newly diagnosed patients developing hypertension (OR=1.76), coronary artery disease (OR=1.65), heart failure (OR=1.76), stroke (OR=1.82) and peripheral arterial disease (OR=1.71).

Conclusions: Statins were used by the majority of the patients and improved the serum lipid levels. However, the improved lipid measures were still indicative of dyslipidemia, with the absence of complete resolution of the condition. Hypertension, coronary artery disease, stroke and peripheral arterial disease are risk factors and frequently associated comorbidities with an unclear causal relationship.

Keywords: Cholesterol, Cardiovascular disease, Dyslipidemia, Hypercholesterolemia, Triglycerides

INTRODUCTION

Dyslipidemia is a metabolic disorder characterized by an imbalance in serum lipid levels, presenting an imbalance of blood lipids including low levels of high-density lipoprotein cholesterol (HDL-C) high levels of low-density lipoprotein cholesterol (LDL-C), total cholesterol and triglycerides (TG). The underlying mechanisms responsible for altered lipid levels can be genetic, environmental or behavioral. Primary dyslipidemia or familial hypertriglyceridemia (high TG levels) is caused

due to variants within the lipoprotein lipase or apo C-ll gene, disrupting the degradation of TGs and increasing their serum level concentration.

Similarly, mutations in the lipid regulatory mechanisms translate to an impaired metabolic pathway, increasing the levels of lipids in the body.^{2,3} On the other hand, secondary dyslipidemia occurs due to acquired risk factors such as lifestyle modifications and existing comorbidities.¹ Certain factors increase the risk of dyslipidemia such as the absence of regular excercise, high-fat dietary intake,

diabetes, obesity, chronic renal conditions, hepatic conditions, hypothyroidism, smoking and alcohol and drug abuse.⁴ Dyslipidemia is a major risk factor, contributing to the pathogenesis of cardiovascular diseases (CVD), type 2 diabetes mellitus (T2DM) and atherosclerosis.⁵

However, there is a lack of defined symptoms of dyslipidemia, commonly overlapping the clinical manifestations of other conditions such as elevated blood pressure, diabetes, increased weight and obesity and smoking, making it difficult to accurately assess dyslipidemia. This challenge complicates the evaluation of its true prevalence and the mortality associated with the condition.¹

Among the Indian population, CVD accounts for 48% of the deaths caused by non-communicable diseases, with CVD developing in Indian patients at a much earlier age compared to those in Western countries.⁶ The Global Burden of Disease reports an unexpected trend, highlighting a rise in cardiovascular-related mortality in developing regions such as India and Africa, while a decline in cardiac deaths is observed in Western countries.⁷

Dyslipidemia is a significant predictor and modifiable risk factor for CVDs. An imbalance in blood lipid profiles can significantly elevate the risk of coronary artery disease (CAD) and CVD. Evidence from American college of cardiology/American heart association (ACC/AHA) suggests that in 56% of coronary heart disease (CHD) patients, dyslipidemia is a predominant causative factor. 10

A strong correlation has been established between dyslipidemia and the global prevalence of cardiovascular disease (CVD), with South Asian populations being particularly affected. 11 Effective management of dyslipidemia, a modifiable predictor for both CVD and CAD, is hypothesized to significantly reduce cardiac-related morbidity and mortality. 5,12

Despite this potential, dyslipidemia remains largely undiagnosed and inadequately controlled among Indian patients. Consequently, the burden of CVD in this population continues to rise due to the underdiagnosis and insufficient management of the condition.

The demographic profile of dyslipidemia in India is influenced by multiple factors, including age, gender, geographic distribution, socioeconomic status, lifestyle behaviors and genetic predispositions. ¹³ Approximately 20% of the adolescent population presents with dyslipidemia.

Among these, the risk of dyslipidemia increases in obese adolescents that demonstrate a prevalence of 42%. 14 Dyslipidemia emerging during early developmental stages tends to persist throughout life, substantially elevating the risk of premature CVD. Thus, early identification and timely intervention of dyslipidemia have the potential to enhance cardiovascular health and mitigate the associated

morbidity and mortality burden.¹ National studies, such as the ICMR-INDIAB provide valuable insights into the prevalence of dyslipidemia across different states, urbanrural divides and socioeconomic strata.¹⁵ However, it is necessary to understand lipid profiles, risk factors, comorbidities and treatment patterns among Indian patients with dyslipidemia.

The primary objective of the UNICORN study was to determine the risk factors for dyslipidemia among different demographic groups. The secondary objectives of the study were to identify any correlation between dyslipidemia and other cardiovascular risk factors and assess the efficacy of the prevalent therapies in managing dyslipidemia.

Using real-world data, the study aims to provide substantial evidence for developing evidence-based interventions, policies and public health strategies targeted at mitigating the burden of dyslipidemia and its associated cardiovascular risks within India's diverse population.

METHODS

Study design

The UNICORN study is a retrospective cross-sectional design.

Study duration

The study period was from May 2024 to September 2024.

The data was retrospectively collected from multiple tertiary care centres across India (centres-1230, number of patients N=9998) including hospitals, clinics and other healthcare institutes. The data was extracted from the existing medical records by general physicians, diabetologists or cardiologists.

Inclusion criteria

Inclusion criteria are patients with a diagnosis of dyslipidaemia with data on existing comorbidity and management approaches and patients receiving pharmacological agents for managing the same.

Exclusion criteria

Patients with alternative diagnoses or missing data for any field were excluded. Patient selection was at the discretion of the treating physician. No additional clinical evaluations or investigations were performed to capture data in this form.

Data collection

The study collected a comprehensive set of variables to facilitate a robust analysis of dyslipidemia and its associated factors. Data was retrieved from the health records by physicians from various institutes in India. Standard format for data collection was utilized. The data collected included 1) Demographic variables age, gender, body mass index (BMI) and the presence of comorbid conditions, 2) Lipid profile parameters, 3) Behavioral variables (risk factors), smoking status, alcohol consumption and dietary habits and 4) Healthcare-related variables, treatment history of medication, duration of dyslipidemia (newly-diagnosed or prevalent dyslipidemia) and serum lipid profiles.

Ethical consideration

The study adheres to the Indian Council of Medical Research (ICMR) "Ethical Guidelines for Biomedical Research on Human Participants" and has been classified as presenting less than minimal risk. Appropriate approval was obtained from an independent Ethics Committee (EC). As the study employs a retrospective design with data without patient identifiers, a waiver of informed consent was sought from the Ethics Committee. Throughout the study, data confidentiality was maintained, with limiting access only to necessary investigators and regulatory authorities.

Data analysis

Data from all participating centers was compiled by the study sponsor, Alkem Laboratories Ltd. The statistical methods employed in the analysis included descriptive statistics to summarize cohort characteristics and lipid profiles, using means, standard deviations and prevalence rates.

Associations between BMI categories and lipid profiles were assessed through hypothesis testing, with statistical significance determined using p values (p<0.05). The relationship between lipid profiles and comorbidities was evaluated via comparative analysis, identifying significant differences based on p values. Odds ratios (OR) were calculated to evaluate the likelihood of comorbid conditions among newly diagnosed versus non-newly diagnosed cases. The impact of treatment on lipid profiles was examined using paired t tests to compare pretreatment and post-treatment levels of total cholesterol, LDL-C and TGs, with significance assessed at p<0.05. All statistical analyses were conducted using R and SAS software.

RESULTS

Patient demographics

The study cohort, comprising 9,998 individuals diagnosed with dyslipidemia, has an average age of 57 years (SD=10.7). The cohort is predominantly male (69.44%), with 67.33% of cases classified as newly diagnosed, indicating a considerable proportion of recent diagnoses. The lifestyle behaviours include smoking in 21.58% and alcohol consumption in 18.26% of the population. The

prevalence of comorbidities is notable, with 71.71% having hypertension, 38.55% having CAD and 7.27% classified as obese (Table 1).

Medication and dietary practices

In the cohort of dyslipidemia patients, a significant majority are on statins, with 99.19% of individuals utilizing these medications. Fibrates are used by 18.88% of the patients, while 8.85% are on ezetimibe. Bempedoic acid is prescribed to a smaller fraction, 3.06% of the cohort (Table 2). Regarding dietary practices, 61.61% of patients adhere to a regular diet. In contrast, others follow specialized dietary regimens. Among these, 44.36% engage in a low-carb diet and 24.15% practice intermittent fasting (Table 2).

BMI and lipid profiles

The analysis of BMI categories reveals significant associations with lipid profiles (p<0.05). Individuals classified as underweight (BMI<18.5 kg/m²) exhibit elevated total cholesterol levels, averaging 266.6 mg/dl. In contrast, those categorized as overweight (BMI: 23.0-24.9 kg/m²) have lower total cholesterol levels, averaging 183.7 mg/dl. HDL-C levels also vary across BMI categories, with higher HDL-C levels observed in obese individuals (52.9 mg/dl) compared to those within the normal BMI range (51.7 mg/dl) (Table 3).

Comorbidities and lipid profiles

The relationship between lipid profiles and comorbidities demonstrates significant associations between dyslipidemia and conditions such as hypertension, CAD, heart failure, stroke and peripheral arterial disease (PAD) (Table 4). Specifically, individuals with hypertension demonstrated significantly elevated total cholesterol (239.5 mg/dl) and TG levels (211.7 mg/dl) compared to those without hypertension (p=0.001).

Patients with CAD presented with higher total cholesterol and LDL-C levels relative to those without CAD. While heart failure is associated with substantial differences in LDL-C and HDL-C levels, total cholesterol levels remain relatively stable across groups with and without heart failure. These findings highlight the complex interplay between dyslipidemia and cardiovascular health, especially in patients with pre-existing comorbid conditions.

Odds ratios for newly diagnosed cases

Odds ratios (OR) were calculated to evaluate the likelihood of comorbid conditions among newly diagnosed versus non-newly diagnosed dyslipidemia cases (Table 5). Newly diagnosed individuals are 1.76 times more likely to have hypertension, 1.65 times more likely to have CAD and 1.76 times more likely to present with heart failure compared to their non-newly diagnosed counterparts. The

odds ratio for stroke is even higher, with newly diagnosed patients nearly twice as likely to have a history of stroke (OR=1.82). Additionally, newly diagnosed dyslipidemic patients are 1.71 times more likely to present with PAD.

Impact of treatment on lipid profiles

Paired t-tests assessing the effect of drug and dietary recommendations on lipid profiles revealed significant reductions in total cholesterol (252.2 \pm 65.85 mg/dl pretreatment vs 195.4 \pm 56.35 mg/dl post-treatment, p<0.001) and LDL-C (158.4 \pm 47.80 mg/dl pre-treatment vs. 121.3 \pm 41.18 mg/dl post-treatment, p<0.001).

TG levels also significantly decreased (216.6±75.84 mg/dl pre-treatment vs. 178.1±61.19 mg/dl post-treatment, p<0.001). No significant changes were observed in HDL-C levels (Table 6).

Table 1: Patient demographic details.

	N	%
Age in mean, SD (in years)	57.0	10.7
Gender		
Male	6942	69.44
Female	3055	30.56
Height in mean, SD (cm)	163.0	9.3
Weight in mean, SD (kg)	74.9	19.8
Newly diagnosed		
Yes	6728	67.33
No	3269	32.72
Smoking status		
Yes	2156	21.58
No	7841	78.50
Alcohol status		
Yes	1823	18.26
No	8174	81.86
Tobacco use		
Yes	1204	12.06
No	8793	88.09
Family history		
Yes	2968	29.74
No	7029	70.44
Comorbidities		
Hypertension	7155	71.71
CAD	3846	38.55
Heart failure	2212	22.18
Stroke	2345	23.51
Peripheral arterial disease	2031	20.36
Obesity		
Obese (>25 kg/m2)	725	7.27
Over weight (23.0 -24.9 kg/m2)	3581	35.92
Normal BMI (18.5-22.9 kg/m2)	4478	44.92
Underweight (<18.5 kg/m2)	1213	12.17

Table 2: Prescribed drug for dyslipidemia and diet.

Prescribed drug for dyslipidemia	N	%
Statins	9858	99.19
Fibrates	1876	18.88
Ezetimibe	879	8.85
Bempedoic acid	304	3.06
Nicotinic Acid	274	2.76
Bile acid sequestrants (BAS)	257	2.59

Continued.

Prescribed drug for dyslipidemia	N	0/0
Dietary advice		
Regular diet	6118	61.61
Low-carb diets	4405	44.36
The DASH Diet	2010	20.24
Weight watchers	2545	25.63
Intermittent fasting	2397	24.15
Information not available	1474	14.85
Any other	79	0.80

Table 3: Levels of BMI and their association with lipid profile.

Total cholestero (mg/dl)		ol	LDL (mg/dl)			HDL (mg/dl)			(TG) (mg/dl)				
BIVII IN	Mean	SD	P value	Mean	SD	P value	Mean	SD	P value	Mean	SD	P value	
Obese (>25 kg/m2)	3581	221.6	68.9		139.0	53.0		52.9	38.1		195.0	83.8	
Overweight (23.0 -24.9 kg/m2)	725	183.7	74.7		134.6	48.9		64.4	34.0		169.9	92.0	
Normal BMI (18.5-22.9 kg/m2)	4475	245.3	58.3	0.001*	152.5	56.3	0.001*	51.7	38.3	0.001*	218.7	80.6	0.001*
Underweight (<18.5 kg/m2)	1209	266.6	56.9		160.0	42.6		47.5	27.8		228.7	80.1	

^{*}P value significant at 95% cI computed using One-way ANOVA test

Table 4: Comorbidities and their association with lipid profile.

		Total c (mg/dl)	holester	terol LDL (mg/dl)			HDL (mg/dl)			TG (mg/dl)			
		Mean	SD	P value	Mean	SD	P value	Mean	SD	P value	Mean	SD	P value
Hypertension													
Yes	7155	239.5	61.1	0.001*	150.4	51	0.001*	47.2	31.2	0.001*	211.7	81.7	0.001*
No	2842	223.4	77.8	•	139.4	59.5		66.1	45.9		198.4	89.3	
CAD													
Yes	3846	240.5	62.3	0.001*	153	52.5	0.001*	48.1	37.6	0.001*	212.7	78.1	0.001*
No	6151	231.4	69		143.7	54.3		55.3	36.3		204.9	87.6	
Heart failure													
Yes	2212	234	65.8	0.475	151.6	56.8	0.01*	54.7	45.5	0.01*	210.1	82.5	0.162
No	7785	235.2	66.9		146	52.8		51.9	34.2		207.3	84.6	
Stroke													
Yes	2345	230.3	49.9	0.001*	152.4	55.1	0.001*	52.5	44.8	0.913	207.1	85.4	0.589
No	7652	236.3	70.9		145.7	53.2		52.6	34.3		208.2	83.8	
PAD													
Yes	2031	238.1	68.6	0.02*	154	53	0.001*	55.3	47.4	0.001*	215	87.8	0.001*
No	7966	234.1	66.1		145.6	53.8		51.9	33.8		206.1	83.1	

^{*}P value significant at 95% CI computed using independent samples t-test

Table 5: Odds ratios for newly diagnosed cases with respect to comorbidities.

Condition	Newly diagnosed	Yes	No	Total	Odds ratio
Hypertension	Yes	5077	2078	7155	1.76
	No	1651	1191	2842	
	Total	6728	3269	9997	
CAD	Yes	2843	1003	3846	1.65
	No	3885	2266	6151	

Continued.

Condition	Newly diagnosed	Yes	No	Total	Odds ratio
	Total	6728	3269	9997	
Heart failure	Yes	1689	523	2212	1.76
	No	5039	2746	7785	
	Total	6728	3269	9997	
Stroke	Yes	1799	546	2345	1.82
	No	4929	2723	7652	
	Total	6728	3269	9997	
PAD	Yes	1546	485	2031	1.71
	No	5182	2784	7966	
	Total	6728	3269	9997	

Table 6: Pre-post analysis of lipid levels in patients receiving dyslipidemia management.

Markers	Timepoint	N	Mean	SD	P value
Total cholesterol (mg/dl)	Pre	4096	252.2152	65.85096	0.001*
	Post	4096	195.437	56.34536	
LDL (mg/dl)	Pre	4096	158.3626	47.80264	0.001*
	Post	4096	121.2845	41.17703	
HDL (mg/dl)	Pre	4096	47.7907	35.27187	0.52
	Post	4096	47.9923	25.84578	
TG (mg/dl)	Pre	4097	216.6161	75.84108	0.001*
	Post	4097	178.1227	61.18718	

^{*}P value significant at 95% CI computed using paired t test

DISCUSSION

A significant proportion of the study population had preexisting conditions such as CAD, hypertension and obesity. Over 99% of patients were undergoing statin therapy, while the majority of the patient did not implement any dietary changes. Treatment following medication with or without diet effectively reduced levels of LDL-C, TGs and total cholesterol. However, total cholesterol levels were elevated in patients with hypertension or CAD.

An uncommon correlation between BMI and dyslipidemia was identified. Obese and overweight patients exhibited higher HDL-C and lower LDL-C levels compared to those with normal or underweight status. Additionally, newly diagnosed individuals were more likely to have existing comorbidities than those with prevalent diagnoses.

The present study cohort demonstrated an unusual lipidprofile. The majority of the patients had hypercholesterolemia, hypertriglyceridemia and high LDL-C. This is in contrast with other studies where the Asian population with dyslipidemia had low-HDL-C hypertriglyceridemia. ^{16,17} While most of the patients had high levels of HDL-C, the protective effect of HDL-C was not observed in patients. Studies have reported a comparatively lower protective effect of HDL-C against CAD/CVD in Indian patients in comparison with other ethinicites. ^{18,19}

Possibly, due to the high levels of HDL-C among Indians leading to increased cardiac risks.²⁰ An increased

prevalence of HDL-C dysfunction among Indians is largely attributed to the significant proportion of patients with underlying metabolic syndromes.²¹ Consequently, despite elevated HDL-C levels, the current study reports a high incidence of CAD and heart failure in this population.

Over 70% of the dyslipidemic patients reported high blood pressure. This finding is in alignment with previous literature that has identified dyslipidemia as an independent risk factor for hypertension. Atherosclerosis as a result of dyslipidemia lead to structural abnormalities within the major blood vessels, subsequently reducing vessel elasticity and causing hypertension.

Additionally, endothelial dysfunction causeddue to altered lipid levels impairs nitric oxide synthesis and secretion resulting in abnormal vasomotor activity and thereby raising blood pressure.²⁴ Therefore, dyslipidemia contributes to the development of hypertension.

The mean total cholesterol levels, LDL-C levels and TG levels of obese and overweight patients are lower than those with normal BMI or underweight patients.

Furthermore, HDL-C levels in obese and overweight patients is greater than the HDL-C levels in patients with normal and underweight BMI. This is an uncommon finding as the majority of the literature demonstrates a strong positive relation between BMI and dyslipidemia. 25,26

According to existing literature, dyslipidemia is significantly associated with the BMI of the patients. The

underlying mechanism for the observed phenomenon includes the fast delivery of free fats to the liver from the surrounding adipose tissue storing fats. Furthermore, the pro-inflammatory stage of the patients due to macrophage infiltration and insulin resistance. However, the present patients with obesity and overweight BMI showed less severe dyslipidemia. A plausible explanation is the low proportion of the obese population included in the study. The present study demonstrated an interesting finding revealing that newly diagnosed patients were at a greater risk of developing comorbidities such as hypertension, CAD, heart failure, PAD and stroke compared to their newly diagnosed counterparts.

This indicates that newly diagnosed dyslipidemia may already be associated with or contribute to the early development of these cardiovascular conditions, likely due to prolonged undiagnosed hyperlipidemia. Consequently, newly diagnosed dyslipidemia often presents alongside major cardiovascular comorbidities. Early and aggressive management is essential to reduce the risk of further vascular complications.

In the present study, a significant majority were on statins to regulate their lipid profile, which is in alignment with the guidelines outlined by the Lipid Association of India (LAI) and AHA/ACC. ^{30,31} The guidelines recommends statins as the first line of treatment for hypercholesterolemia to preventatherosclerotic CVD.

However, previous pharmacokinetic studies have consistently demonstrated that the Asian population exhibits a stronger response to statin therapy compared to its Western counterparts. ^{17,32,33} Caucasians often require higher statin dosages to achieve the same pharmacological effect as Asian individuals. ^{17,32,33}

The enhanced response to statins in Asian populations potentially increases the risk of drug-related adverse effects. Myalgia, neurological conditions, elevated serum glycemic profile and cognitive changes are among the commonly reported side effects associated with the use of statins.³⁴ As majority of the data is derived from the Western regions with low proportion of the Indian population, careful monitoring for these adverse events is essential when prescribing statins in India.

Furthermore, a significantly lower proportion of the study cohort followed a diet to improve dyslipidemia. Among those following dietary patterns, low-carbohydrate and dietary approaches to stop hypertension (DASH) were the most common. The low adherence to special diets is highly contrasted with existing literature with evidence suggesting a critical role of diet intake in dyslipidemia progression and successful outcome in patients with dietary management. ^{15-17,35-37}

The DASH diet is a plant-based diet with reported efficacy in reducing serum TG and LDL-C levels in patients.³⁸ Addisu et al, (2023) reported a positive correlation

between low plant intake and LDL-C dyslipidemia.³⁹ The positive effect of plant intake on serum lipid levels is due to the protective effects of vitamins, minerals and other micronutrients present in high quantities in fruits and vegetables.

These nutritional factors are anti-inflammatory and anti-oxidative which improve insulin sensitivity and hypertension, subsequently alleviating dyslipidemia.³⁹ The impact of low-carbohydrate diet on improving dyslipidemia is highly controversial due to the incorporation of high fats. According to studies low carbohydrate diet decreases LDL-C in obese patients, with the opposite effect in lean patients increasing cholesterol levels.⁴⁰ Furthermore, Mansoor et al, (2016) reported a significant increase in LDL-C levels to post low-carbohydrate practices with an increased risk of CVD.⁴¹

The present study was a retrospective cross-sectional analysis of 9,998 patient health records, which significantly enhanced the sample size and robustness of the findings. Patient selection was based on the discretion of the physicians eliminating bias in the study participants. Furthermore, patients were enrolled from various geographical locations of India, increasing the generalizability of the study results to the Indian population. The UNICORN study provides data for the regulatory bodies to improve the policies and tailor the guidelines specific to Indian patients.

The study has certain limitations, first, the retrospective nature of the study limits the identification of the causal factors responsible for dyslipidemia due to the absence of a comparator arm. Second, the study does not account for patient characteristics such as socioeconomic status, education level or geographic location, which may influence patient outcomes and treatment adherence. Third, age stratification could have been beneficial for identifying at-risk populations within India.

CONCLUSION

Hypertension, coronary artery disease, stroke and PAD are frequently associated comorbidities with an unclear causal relationship. Furthermore, newly-diagnosed patients are at a significantly higher risk of comorbidities. Therefore, effective treatment strategies are required for early prevention.

As statins are widely accepted for managing dyslipidemia, there exists a need for improved statins with low side effects and a higher capacity to reduce cholesterol. The UNICORN study provides preliminary evidence for identification of risk factors and timely management of dyslipidemia in the context of the Indian population. Further randomized clinical trials are required to validate the findings, refine risk assessment models and develop evidence-based guidelines for the prevention, early detection and management of dyslipidemia and its associated cardiovascular risks.

Funding: No funding sources Conflict of interest: None declared

Ethical approval: The study was approved by the

Institutional Ethics Committee

REFERENCES

- Pappan N, Awosika A, Rehman A. Dyslipidemia -StatPearls-NCBI Bookshelf. StatPearls Publ. 2024.
- 2. Mozaffarian D, Benjamin EJ, Go AS. Heart disease and stroke statistics-2016 Update: a report from the american heart association. Circulation. 2016;133(4):38-360.
- 3. Defesche JC, Gidding SS, Harada-Shiba M, Hegele RA, Santos RD, Wierzbicki AS. Familial hypercholesterolaemia. Nat Rev Dis Primer. 2017;3:17093.
- Saavedra A, Rodrigues E, Carvalho D. Dyslipidemia Secondary to Hypothyroidism and Cholestasis. Acta Med Port. 2020;33(3):204-7.
- Al Quran TM, Bataineh ZA, Al-Mistarehi AH. Prevalence and pattern of dyslipidemia and its associated factors among patients with type 2 diabetes mellitus in Jordan: a cross-sectional study. Int J Gen Med. 2022;15:7669-83.
- Ke C, Gupta R, Xavier D. Divergent trends in ischaemic heart disease and stroke mortality in India from 2000 to 2015: a nationally representative mortality study. Lancet Glob Health. 2018;6(8):e914-23.
- Kiran G, Mohan I, Kaur M, Ahuja S, Gupta S, Gupta R. Escalating ischemic heart disease burden among women in India: Insights from GBD, NCDRisC and NFHS reports. Am J Prev Cardiol. 2020;2:100035.
- Carey VJ, Bishop L, Laranjo N, Harshfield BJ, Kwiat C, Sacks FM. Contribution of high plasma triglycerides and low high-density lipoprotein cholesterol to residual risk of coronary heart disease after establishment of low-density lipoprotein cholesterol control. Am J Cardiol. 2010;106(6):757-63.
- 9. Bloomgarden ZT. American diabetes association annual meeting, 1999: more on cardiovascular disease. Diabetes Care. 2000;23(6):845-52.
- 10. Grundy SM, Stone NJ, Bailey AL. AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/A GS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: a report of the American college of cardiology/American heart association task force on clinical practice guidelines. Circulation. 2019;139:25.
- 11. Reese JA, Roman MJ, Deen JF. Subclinical atherosclerosis in adolescents and young adults and the risk of cardiovascular disease: the strong heart family study (SHFS). Nutr Metab Cardiovasc Dis. 2022;32(8):1863-71.
- 12. Costa J, Borges M, David C, Carneiro AV. Efficacy of lipid lowering drug treatment for diabetic and non-diabetic patients: meta-analysis of randomised controlled trials. BMJ. 2006;332(7550):1115-24.

- Sharma S, Gaur K, Gupta R. Trends in epidemiology of dyslipidemias in India. Indian Heart J. 2024;76:20-8
- Gujral J, Gupta J. Pediatric Dyslipidemia StatPearls NCBI Bookshelf. StatPearls Publ. Published online 2024.
- 15. Anjana RM, Unnikrishnan R, Deepa M. Metabolic non-communicable disease health report of India: the ICMR-INDIAB national cross-sectional study (ICMR-INDIAB-17). Lancet Diabetes Endocrinol. 2023;11(7):474-89.
- 16. Misra A, Shrivastava U. Obesity and dyslipidemia in South Asians. Nutrients. 2013;5(7):2708-33.
- 17. Misra S, Lyngdoh T, Mulchandani R. Guidelines for dyslipidemia management in India: A review of the current scenario and gaps in research. Indian Heart J. 2022;74(5):341-50.
- 18. Bilen O, Kamal A, Virani SS. Lipoprotein abnormalities in South Asians and its association with cardiovascular disease: Current state and future directions. World J Cardiol. 2016;8(3):247.
- 19. Chow CK, McQuillan B, Raju PK. Greater adverse effects of cholesterol and diabetes on carotid intimamedia thickness in South Asian Indians: Comparison of risk factor-IMT associations in two population-based surveys. Atherosclerosis. 2008;199(1):116-22.
- 20. Dodani S, Kaur R, Reddy S, Reed GL, Navab M, George V. Can dysfunctional HDL explain high coronary artery disease risk in South Asians? Int J Cardiol. 2008;129(1):125-32.
- 21. Forouhi NG, Sattar N, Tillin T, McKeigue PM, Chaturvedi N. Do known risk factors explain the higher coronary heart disease mortality in South Asian compared with European men? Prospective follow-up of the Southall and Brent studies, UK. Diabetologia. 2006;49(11):2580-8.
- 22. Wyszyńska J, Łuszczki E, Sobek G, Mazur A, Dereń K. Association and risk factors for hypertension and dyslipidemia in young adults from Poland. Int J Environ Res Public Health. 2023;20(2):982.
- 23. Oparil S, Zaman MA, Calhoun DA. Pathogenesis of hypertension. Ann Intern Med. 2003;139(9):761.
- 24. Sesso HD. A prospective study of plasma lipid levels and hypertension in Women. Arch Intern Med. 2005;165(20):2420.
- 25. Kuwabara M, Kuwabara R, Niwa K. Different risk for hypertension, diabetes, dyslipidemia and hyperuricemia according to level of body mass index in Japanese and American subjects. Nutrients. 2018;10(8):1011.
- 26. Zheng C, Liu Y, Xu C. Association between obesity and the prevalence of dyslipidemia in middle-aged and older people: an observational study. Sci Rep. 2024;14(1):11974.
- 27. Klop B, Elte J, Cabezas M. Dyslipidemia in Obesity: Mechanisms and Potential Targets. Nutrients. 2013;5(4):1218-40.
- 28. Xiao C, Dash S, Morgantini C, Hegele RA, Lewis GF. Pharmacological targeting of the atherogenic dyslipidemia complex: the next frontier in CVD

- prevention beyond lowering LDL cholesterol. Diabetes. 2016;65(7):1767-78.
- 29. Björnson E, Adiels M, Taskinen MR, Borén J. Kinetics of plasma triglycerides in abdominal obesity. Curr Opin Lipidol. 2017;28(1):11-8.
- Arnett DK, Blumenthal RS, Albert MA. 2019 ACC/AHA Guideline on the primary prevention of cardiovascular disease: a report of the American college of cardiology/American heart association task force on clinical practice guidelines. Circulation. 2019;140:11.
- 31. Puri R, Bansal M, Mehta V. Lipid Association of India 2023 update on cardiovascular risk assessment and lipid management in Indian patients: Consensus statement IV. J Clin Lipidol. 2024;18(3):351-73.
- 32. Naito R, Miyauchi K, Daida H. Racial differences in the cholesterol-lowering effect of statin. J Atheroscler Thromb. 2017;24(1):19-25.
- 33. Rajesh C, Yesudas A, Mahadevan L. Prevalence of genetic variants associated with cardiovascular disease risk and drug response in the Southern Indian population of Kerala. Indian J Hum Genet. 2014;20(2):175.
- 34. Mulchandani R, Lyngdoh T, Kakkar AK. Statin use and safety concerns: an overview of the past, present and the future. Expert Opin Drug Saf. 2020;19(8):1011-24.
- 35. Khadke S, Mandave P, Kuvalekar A, Pandit V, Karandikar M, Mantri N. Synergistic effect of omega-3 fatty acids and oral-hypoglycemic drug on lipid normalization through modulation of hepatic gene expression in high fat diet with low streptozotocin-induced diabetic rats. Nutrients. 2020;12(12):3652.

- Enani S, Bahijri S, Malibary M. The association between dyslipidemia, dietary habits and other lifestyle indicators among non-diabetic attendees of primary health care centers in Jeddah, Saudi Arabia. Nutrients. 2020;12(8):2441.
- 37. Tan LJ, Kim SA, Shin S. Association between three low-carbohydrate diet scores and lipid metabolism among Chinese adults. Nutrients. 2020;12(5):1307.
- 38. Sahebkar A, Heidari Z, Kiani Z. The efficacy of dietary approaches to stop hypertension (DASH) diet on lipid profile: A systematic review and meta-analysis of clinical controlled trials. Curr Med Chem. 2023;31:467-9.
- Addisu B, Bekele S, Wube TB, Hirigo AT, Cheneke W. Dyslipidemia and its associated factors among adult cardiac patients at Ambo university referral hospital, Oromia region, west Ethiopia. BMC Cardiovasc Disord. 2023;23(1):321.
- Soto-Mota A, Flores-Jurado Y, Norwitz NG. Increased low-density lipoprotein cholesterol on a low-carbohydrate diet in adults with normal but not high body weight: A meta-analysis. Am J Clin Nutr. 2024;119(3):740-7.
- 41. Mansoor N, Vinknes KJ, Veierød MB, Retterstøl K. Low-carbohydrate diets increase LDL-cholesterol and thereby indicate increased risk of CVD. Br J Nutr. 2016;115(12):2264-6.

Cite this article as: Mayabhate M, Sharma A. Unravelling the risk factors and treatment patterns of dyslipidemia in India (UNICORN study). Int J Res Med Sci 2025;13:164-72.