Section: Cardiology

Original Research Article

PREVALENCE OF FAMILIAL DYSLIPIDEMIAS IN PATIENTS WITH PREMATURE CORONARY ARTERY DISEASE IN NORTHERN INDIA AND ITS CORRELATION WITH ANGIOGRAPHIC SEVERITY OF DISEASE

Mohammed Sheeraz Alam[1], Malik Mohd Azharuddin[2], Sameera Khan[3], Mohd Shafique Alam[4]

[1]*Resident, Department of Cardiology, JN Medical College, AMU, Aligarh, Uttar Pradesh India.*
[2]*Professor, Department of Cardiology, JN Medical College, AMU, Aligarh, Uttar Pradesh India.*
[3]*PG Resident, Department of Anaesthesiology, Sharda School of Medical Sciences and Research, Greater Noida, Uttar Pradesh, India.*
[4]*Resident, Department of Cardiology, JN Medical College, AMU, Aligarh, Uttar Pradesh India.*

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Corresponding Author:
Dr. Mohammed Sheeraz Alam,
Resident, Department of Cardiology, JN Medical College, AMU, Aligarh, Uttar Pradesh India.
Email: sheerazalam08@gmail.com

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ABSTRACT

Background: Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide, with its burden increasingly significant in India. Familial dyslipidemias, particularly familial hypercholesterolemia (FH), play a crucial role in the development of premature CAD. This study investigates the prevalence of familial dyslipidemias in young patients presenting with premature CAD and its correlation with the angiographic severity of the disease in a Northern Indian population. **Objectives:** To assess the prevalence of familial dyslipidemias in young patients (≤45 years) with premature CAD and to evaluate the correlation between lipid profile abnormalities and angiographic findings in this cohort.

Materials and Methods: This observational, cross-sectional study was conducted at Jawaharlal Nehru Medical College and Hospital, Aligarh, from October 2022 to October 2024. The study included 100 consecutive young patients with a provisional diagnosis of acute or chronic coronary syndrome and angiographically confirmed CAD. A total of 203 serum lipid profile samples were collected from patients and their family members to identify familial dyslipidemias. Data on demographic, clinical, biochemical, and angiographic parameters were analyzed. The Dutch Lipid Clinic Network (DLCN) criteria were used to identify FH, categorizing patients into definite, probable, or unlikely FH.

Results: Of the 100 patients, 22% were diagnosed with "potential FH" (combining definite and probable FH groups). Males were predominantly affected (81%), and the mean age of the study cohort was 38.42 ± 3.5 years for males and 39.19 ± 4.1 years for females. In 22% patient categorized as Potential FH group the mean LDL-C was 234.45 ± 56.99mg/dl which has significant p value (p value <0.001) when compared with mean LDL-C of Unlikely FH group. Angiographic analysis revealed that 42% had double-vessel disease (DVD), while 41% had single-vessel disease (SVD). In cascade screening 103 first generation relatives were identified and their Serum lipid were checked.

Conclusion: Familial dyslipidemias, particularly FH, are prevalent among young patients with premature CAD in Northern India and are associated with severe angiographic findings. Early detection through lipid screening and genetic counseling, combined with aggressive lipid-lowering therapies and lifestyle interventions, is critical to reducing the burden of premature CAD in this population.

Keywords: Coronary artery disease, familial dyslipidemias, premature CAD, familial hypercholesterolemia, angiographic severity, lipid profile, Northern India.

INTRODUCTION

Global Burden of Coronary Artery Disease (CAD)

Coronary artery disease (CAD) remains the leading cause of mortality and morbidity worldwide. According to the World Health Organization (WHO), CAD was responsible for approximately 17.9 million deaths in 2019, accounting for 31% of all global deaths (WHO, 2020). The prevalence of CAD is rising in low- and middle-income countries (LMICs) due to urbanization, sedentary lifestyles, and increasing rates of risk factors such as hypertension, diabetes, and dyslipidemia. The disease is characterized by the narrowing of coronary arteries due to atherosclerotic plaque build-up, which can lead to myocardial ischemia, infarction, and death.[1]

Although CAD rates have plateaued or declined in high-income countries due to advancements in prevention and treatment, LMICs face a rising epidemic of CAD due to economic and lifestyle transitions.[2] South Asia, including India, has emerged as a hotspot for CAD prevalence, with unique genetic predispositions and lifestyle risk factors contributing to this burden.[3,4]

2. Pathophysiology and Risk Factors

CAD is primarily driven by atherosclerosis, a process involving lipid accumulation, inflammation, and plaque formation in arterial walls. Low-density lipoprotein cholesterol (LDL-C) plays a central role in this process, and its reduction is a key target for CAD prevention and treatment[5]. Emerging research highlights the role of other factors, such as lipoprotein(a) and triglycerides, in CAD pathophysiology, particularly in populations with distinct lipid profiles.[6]

3. CAD in India

Epidemiological Trends

India has seen a dramatic rise in CAD prevalence over the past two decades, with urban areas reporting a prevalence of 9-10% and rural areas 4-5%[9]. This trend is attributed to urbanization, dietary changes, and increasing prevalence of hypertension, diabetes, and dyslipidemia.[4]

The "Asian Indian phenotype," characterized by high triglycerides, low HDL cholesterol, and small dense LDL particles, predisposes Indians to premature CAD.[3] This phenotype is compounded by lifestyle factors, including physical inactivity, poor dietary habits, and high levels of psychosocial stress.[7]

Young CAD

Premature CAD, defined as onset before 45 years of age, is more prevalent in India compared to Western countries[8]. Risk factors include smoking, central obesity, and familial dyslipidemias. Studies have shown that young CAD patients often present with more severe forms of the disease, such as triple-vessel disease (TVD) and high coronary artery calcium scores.[10]

4. Familial Dyslipidemias

Overview

Familial dyslipidemias are genetic conditions that result in abnormal lipid levels, significantly increasing the risk of early-onset CAD. Familial hypercholesterolemia (FH), familial combined hyperlipidemia (FCH), and familial hypoalphalipoproteinemia are the most studied conditions in this category.[11]

Familial Hypercholesterolemia (FH)

FH is an autosomal dominant disorder characterized by elevated LDL-C levels from birth. It is classified into homozygous FH (HoFH) and heterozygous FH (HeFH), with HoFH presenting more severe manifestations. The prevalence of FH is estimated at 1 in 200 to 1 in 500 for HeFH, making it one of the most common genetic disorders.[12]

Studies in India have shown that FH is underdiagnosed and undertreated, with many individuals presenting with myocardial infarction as the first manifestation of the disease.[9] The Dutch Lipid Clinic Network (DLCN) criteria and genetic testing are the standard diagnostic tools for FH.

Familial Combined Hyperlipidemia (FCH)

FCH is a polygenic disorder characterized by elevated LDL-C, triglycerides, and apolipoprotein B levels. It is associated with an increased risk of CAD and often overlaps with metabolic syndrome.[13]

5. Diagnostic Approaches

Lipid Screening

Lipid profile testing, including LDL-C, HDL-C, and triglycerides, is the cornerstone for diagnosing dyslipidemias. Guidelines recommend universal screening for lipid disorders in adults and targeted screening in children with a family history of premature CAD.[14]

Genetic Testing

Genetic testing for mutations in LDL receptor, APOB, and PCSK9 genes confirms FH diagnosis. However, its use in India is limited due to high costs and lack of accessibility.[15]

Clinical Scoring Systems

Scoring systems like the DLCN criteria provide a structured approach for diagnosing FH. A score of ≥6 indicates probable or definite FH, warranting aggressive lipid-lowering therapy.[16]

6. Treatment Strategies

Lipid-Lowering Therapies

Statins are the first-line therapy for reducing LDL-C levels and preventing CAD. High-intensity statins, combined with ezetimibe or PCSK9 inhibitors, achieve significant LDL-C reductions and improve cardiovascular outcomes.[17]

Emerging Therapies

Novel treatments, such as inclisiran (a small interfering RNA targeting PCSK9) and bempedoic acid, offer additional LDL-C reduction options for patients intolerant to statins or with severe dyslipidemias.[18]

Lifestyle Modifications

Lifestyle changes, including a heart-healthy diet, regular physical activity, and smoking cessation, are essential adjuncts to pharmacotherapy.[19]

7. Challenges in India

Underdiagnosis and Treatment Gaps

Despite the high burden of CAD and familial dyslipidemias, awareness and screening remain limited in India. Studies show that a significant proportion of individuals with FH remain undiagnosed, and treatment initiation is often delayed.[10]

Healthcare Infrastructure

India's healthcare system faces challenges in providing widespread access to lipid screening and genetic testing. Public health initiatives targeting awareness and early diagnosis are critical to addressing these gaps.[4]

MATERIALS AND METHODS

Study Design and Setting

This observational, cross-sectional, hospital-based study was conducted in the Department of Cardiology, Jawaharlal Nehru Medical College (JNMC), Aligarh Muslim University, Aligarh, India, over a two-year period from October 2022 to October 2024. The study aimed to assess the prevalence of familial dyslipidemias in young patients presenting with premature coronary artery disease (CAD) and to correlate these lipid abnormalities with the angiographic severity of CAD.

Study Population

The study enrolled 100 consecutive young patients aged ≤45 years who presented with symptoms of acute coronary syndrome (ACS) or chronic coronary syndrome (CCS) and were confirmed to have coronary artery disease through angiography. Cascade screening was done among the study population and their first degree relatives were also screening for the presence of Familial Dyslipidemias and an additional 103 family members (parents and siblings) were included to evaluate the hereditary patterns of dyslipidemias.

Inclusion Criteria

1. Patients aged ≤45 years presenting with ACS, including ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI), or chronic coronary syndrome.
2. Patients with angiographically confirmed CAD.
3. Patients and family members willing to provide informed consent.

Exclusion Criteria

1. Patients older than 45 years.
2. Patients or families who declined to provide consent.
3. Patients with secondary causes of dyslipidemia, such as hypothyroidism, nephrotic syndrome, or chronic liver disease.

Sample Size Calculation

The sample size was determined using statistical formulas that considered the expected prevalence of familial hypercholesterolemia (FH) and related lipid abnormalities in young CAD patients. Based on previous studies, a minimum of 100 patients was deemed sufficient to achieve statistical significance.

Data Collection

A structured proforma was used to collect detailed demographic, clinical, and laboratory data. Data collection included the following components:

1. **Demographic Information:**
   - Age, gender, socioeconomic status, and family history of CAD.
2. **Clinical History:**
   - History of smoking, alcohol consumption, physical inactivity, and dietary patterns.
   - Personal and family history of cardiovascular events, including myocardial infarction, sudden cardiac death, and stroke.
3. **Physical Examination:**
   - Anthropometric measurements, including body mass index (BMI) and waist-to-hip ratio.
   - Signs of dyslipidemias, such as xanthomas and corneal arcus.

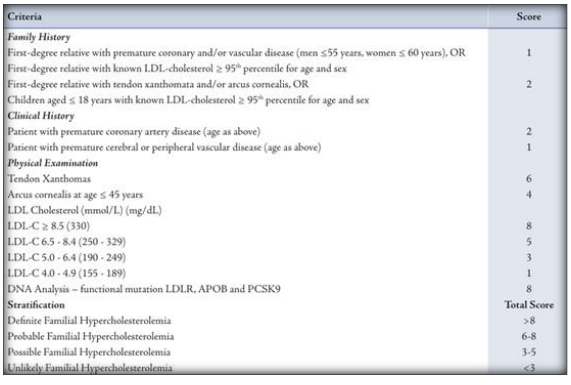
Lipid Profile Assessment

Blood samples were collected after a 12-hour fasting period to measure lipid levels. The lipid parameters assessed included:

1. Total cholesterol (TC): Measured enzymatically using the cholesterol esterase oxidase peroxidase method.
2. Low-density lipoprotein cholesterol (LDL-C): Calculated using the Friedewald formula: LDL-C = TC – (HDL-C + TG/5).
3. High-density lipoprotein cholesterol (HDL-C): Measured by enzymatic precipitation methods.
4. Triglycerides (TG): Determined using a modified enzymatic colorimetric method.

Diagnosis of Familial Dyslipidemias

Familial dyslipidemias were diagnosed using the Dutch Lipid Clinic Network (DLCN) criteria, which stratifies patients into Definite, Probable, or Unlikely FH based on LDL-C levels, family history, clinical signs, and genetic testing.



Criteria	Score
Family History	
First-degree relative with premature coronary and/or vascular disease (men ≤55 years, women ≤ 60 years), OR	1
First-degree relative with known LDL-cholesterol ≥ 95th percentile for age and sex	
First-degree relative with tendon xanthomata and/or arcus cornealis, OR	2
Children aged ≤ 18 years with known LDL-cholesterol ≥ 95th percentile for age and sex	
Clinical History	
Patient with premature coronary artery disease (age as above)	2
Patient with premature cerebral or peripheral vascular disease (age as above)	1
Physical Examination	
Tendon Xanthomas	6
Arcus cornealis at age ≤ 45 years	4
LDL Cholesterol (mmol/L) (mg/dL)	
LDL-C ≥ 8.5 (330)	8
LDL-C 6.5 - 8.4 (250 - 329)	5
LDL-C 5.0 - 6.4 (190 - 249)	3
LDL-C 4.0 - 4.9 (155 - 189)	1
DNA Analysis – functional mutation LDLR, APOB and PCSK9	8
Stratification	**Total Score**
Definite Familial Hypercholesterolemia	>8
Probable Familial Hypercholesterolemia	6-8
Possible Familial Hypercholesterolemia	3-5
Unlikely Familial Hypercholesterolemia	<3

Coronary Angiography

Coronary angiography was performed on all enrolled patients using standard techniques via the

femoral or radial artery approach. Angiographic images were analyzed by two independent interventional cardiologists to minimize subjective bias. The severity of CAD was categorized based on the number of vessels involved and the degree of stenosis:

1. Single-Vessel Disease (SVD): Significant stenosis (≥70% narrowing) in one coronary artery.
2. Double-Vessel Disease (DVD): Significant stenosis in two coronary arteries.
3. Triple-Vessel Disease (TVD): Significant stenosis in three coronary arteries.
4. Left Main Coronary Artery Disease: Significant stenosis (≥50% narrowing) in the left main coronary artery.

Assessment of Angiographic Features

Angiographic parameters evaluated included:

1. Presence of thrombus, bifurcation lesions, and calcifications.
2. Chronic total occlusions (CTOs).
3. Plaque morphology, including soft and calcified plaques.

Pedigree Analysis

Pedigree analysis is a fundamental tool in genetics used to study the inheritance of traits across generations within a family. It provides a visual representation of a family's history and can help determine the mode of inheritance (dominant, recessive, X-linked, etc.) for a particular genetic trait, disorder, or disease

Statistical Analysis: Data were analyzed using SPSS version 25. Continuous variables were expressed as mean ± standard deviation, and categorical variables were presented as percentages. Group comparisons were performed using independent t-tests for continuous variables and chi-square tests for categorical variables. Correlations between lipid levels and angiographic severity were assessed using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

Ethical Considerations

The study was approved by the Institutional Ethics Committee of JNMC, AMU. Written informed consent was obtained from all participants. Confidentiality of patient data was maintained throughout the study.

RESULTS AND DISCUSSIONS

Out of 100 patients enrolled in the study 79 were male. The mean age of males and females enrolled in the study were 38.42 ± 3.5 and 39.19 ± 4.1 years which was similar in other studies22

In our study population the disease substrate was 32% AWMI, 17% IWMI, 38% NSTEMI, 9% CCS and 4% Unstable Angina. On Angiography of the study patients 56% had SVD, 15% DVD, 8% TVD and 21% Non-Obstructive CAD. Among the culprit vessel LAD was most commonly affected vessel (41%) followed by RCA (22%) and LCX (20%).

The study population were categorized as Definite, Probable, Possible and Unlikely FH according to the DLCN Criteria scoring system. 56% subjects were categorized as Unlikely FH (DLCN <3), 22% as Possible FH (DLCN 3-5), 16% as Probable FH (DLCN 5-7) and 6% as Definite FH (DLCN >8). So, 22% study populations were categorized as Potential FH (Definite + Probable FH).

On comparing the Serum lipid profile of different FH groups, the LDL Cholesterol of Definite FH group was 298.2 ± 18.3, for Probable FH LDL-C was 210.6 ± 16.7, for possible FH LDL-C was 127.3 ± 13.2 and for Unlikely FH LDL-C was 98.8 ± 11.1mg/dl which has significant p value (<0.05). However, there were no significant difference in the number of vessels involved as seen in CAG in these different FH groups. There was no significant difference in the Syntax 1 scoring among these FH groups.

Table 1

Lipid Profile	Definite FH (Mean)	Definite FH (SD)	Probable FH (Mean)	Probable FH (SD)	Possible FH (Mean)	Possible FH (SD)	Unlikely FH (Mean)	Unlikely FH (SD)
TC (mg/dl)	355.3	20.5	300.7	18.4	206.4	15.7	177.8	14.3
LDL (mg/dl)	298.2	18.3	210.6	16.7	127.3	13.2	98.8	11.1
HDL (mg/dl)	42.2	5.1	46.5	4.8	50.1	4.5	54.9	4.2
TG (mg/dl)	142.7	15.2	114.6	13.9	150.8	12.4	122.2	10.8

In 22% patient categorized as Potential FH group the mean LDL-C was 234.45 ± 56.99mg/dl which has significant p value (p value <0.001) when compared with mean LDL-C of Unlikely FH group.

Cascade Screening for Familial Hypercholesterolemia

According to the National Institute for Health and Clinical Excellence (NICE) Guidelines 2017, cascade testing using DNA analysis should be performed in first-degree, second-degree, and possibly third-degree relatives of patients diagnosed with Familial Hypercholesterolemia (FH). However, when genetic testing is not available, a non-fasting serum lipid profile can be conducted for family members (parents, siblings, and children) of diagnosed FH patients. This is a simple, economical, and effective tool to identify carriers of FH. Timely intervention, including dietary advice, lifestyle changes, and early initiation of medications (such as

statins and ezetimibe), based on cascade screening, can help prevent premature CAD in the community.

In cascade screening, the ideal age for lipid profile testing in children remains controversial. According to the National Lipid Association Expert Panel on FH, children born into high-risk families, i.e., those with high cholesterol concentrations or a family history of premature CAD, should undergo lipid screening tests starting at age ≥ 2 years14. Screening for lipids in children younger than 2 years is generally not recommended. The National Health guidelines suggest that universal screening is best performed in children between 9 and 11 years of age who are born into high-risk families20,21.

According to the National Lipid Association's key screening recommendations, FH should be considered in children, adolescents, and young adults under 20 years of age if their LDL concentration exceeds 160 mg/dl in lipid screening tests.

This study is unique in that cascade screening was conducted in patients presenting with premature CAD, and to the best of our knowledge, no other study conducted in Northern India has utilized cascade screening to determine the prevalence of FH in the community.

In our study, we had 100 patients presenting with premature CAD. Through the cascade screening method, we created a pedigree chart for their first-degree relatives, which totalled 478 individuals. Out of these, only 103 (21.6%) people agreed to undergo lipid screening, 179 (37.4%) outrightly denied screening and did not give their consent, and the remaining 196 (41.0%) were living in different cities across the country and could not be traced. Therefore, we screened the lipid levels of 103 relatives (parents, siblings, and children), making a total sample size of 203. Among these 103 relatives 5%, 11%, 16% and 68% were assigned as Definite, Probable, Possible and Unlikely FH respectively.

In the complete sample, 74% were male and 26% were female. The distribution of the study population according to the DLCN score showed Definite FH in 5.4%, Probable FH in 13.3%, Possible FH in 19.2%, and Unlikely FH in 62.1%, with a combined 18.7% classified as Potential FH (Definite + Probable FH).

Lipid screening in these 203 individuals (patients and relatives) revealed the following LDL-C levels: Definite FH had an LDL-C level of 282.8 ± 63.60 mg/dl, Probable FH had an LDL-C concentration of 215.1 ± 35.08 mg/dl, Possible FH had an LDL-C level of 152.0 ± 48.49 mg/dl, and Unlikely FH had 100.1 ± 15.77 mg/dl.

These asymptomatic relatives will undergo further detailed physical examinations to identify other signs of hypercholesterolemia. The presence of tendon xanthoma at any age, arcus cornealis before age 45, and xanthelasma before age 25 strongly suggests the possibility of FH.

Table 2: ?

Lipid Profile	Definite FH (Mean)	Definite FH (SD)	Probable FH (Mean)	Probable FH (SD)	Possible FH (Mean)	Possible FH (SD)	Unlikely FH (Mean)	Unlikely FH (SD)
TC (mg/dl)	354.8	36.15	302.4	36.81	233.5	58.12	178.5	17.40
LDL (mg/dl)	282.8	63.60	215.1	35.08	152.0	48.49	100.1	15.77
HDL (mg/dl)	46.8	9.90	46.3	5.11	48.8	8.79	53.0	8.22
TG (mg/dl)	146.3	51.59	112.0	20.82	136.4	68.82	130.2	56.80

We already know the incidence of FH in general population (Approx 4%) but we did not know the incidence of Potential FH in patients with young CAD (22% in our study). Which is found to be much more than the expected. Also we included the screening of the first generation family members in our study which leads to early diagnosis and treatment of people with Familial Hypercholesterolemia which can be largely controlled if diagnosed on time.

Limitations of the Study

- Firstly, this was a single-centre study. As a tertiary care hospital located in Aligarh, we cater to a wide population from western Uttar Pradesh, but our results may not reflect the true prevalence of FH across the entire diverse population of India.
- Secondly, lipid levels were measured at different laboratories, and interlaboratory variations may have biased the accurate estimation of FH prevalence.
- Thirdly, some patients had already taken statins before their lipid levels were measured, and some relatives were on statin therapy during cascade screening. This may have introduced bias in estimating the true prevalence of FH.
- Importantly, genetic testing was not performed in our study to confirm the diagnosis of FH. This limitation could have resulted in a mix of FH cases with inherited dyslipidemias, such as polygenic FH, which is an important differential diagnosis in our patient cohort.
- Lastly, some patients were not aware of their family history of premature CAD which leads to the underestimation of true FH prevalence.

CONCLUSION

Familial dyslipidaemias are significant contributors to the development of premature coronary artery disease (CAD), particularly in populations like Northern India, where genetic predisposition, environmental factors, and lifestyle changes

converge. Various scoring tools are available for the early detection of FH; however, routine screening with simple criteria, such as a family history of premature CAD combined with a pre-treatment LDL cholesterol level of >155 mg/dL and a DLCN criteria score ≥6, can be effectively used. Early identification and aggressive management of these lipid disorders are crucial for reducing the burden of CAD and improving patient outcomes.

Despite having a case of premature CAD in the family, many individuals are still hesitant and resistant to undergo any screening tests. Educating families about the importance of screening for diseases that are easily modifiable and treatable if detected early cannot be underestimated.

If the large burden of atherosclerotic cardiovascular disease associated with FH is to be reduced, diagnosis and treatment must begin early in life. "By diagnosing a case of FH, we do not identify a case but a family at risk.

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