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IMPACT OF DYSLIPIDEMIA ON HEART ATTACK RISK: PATHOPHYSIOLOGY, CLINICAL RELEVANCE AND THERAPEUTIC INSIGHTS

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ABSTRACT

Dyslipidemia, which is the presence of abnormal lipid levels in the blood, is a major risk factor that can be changed for atherosclerotic cardiovascular disease, including heart attacks and strokes. This review looks at the causes and types of dyslipidemia, its connection to heart events, the range of clinical outcomes, diagnostic methods, and both current and new treatment strategies. New guidelines focus on personalized risk assessment and using multiple medications together. Even with significant progress, there are still gaps in effective risk assessment and long-term care. This calls for more research to lessen the global impact of cardiovascular issues. **Objectives:** The purpose of this review is to provide a detailed overview of the epidemiology, causes, and classification of dyslipidemia. Examine its pathophysiology and its connection to cardiovascular disease. It summarizes the clinical signs, diagnostic methods, and complications. And also critically evaluate management strategies and recent improvements in therapy. Identify current research gaps and future directions for better CVD risk reduction. **Methods:** This review adopts a narrative approach to synthesizing evidence from secondary sources. Databases including PubMed, Scopus, Web of Science, and Google Scholar were searched for articles using keywords such as Dyslipidemia, Lipid abnormalities, cardiovascular disease, heart attack, myocardial infarction, diagnosis of dyslipidemia, and lipid - lowering therapy. **Results:** Dyslipidemia affects about 30-40% of adults globally and is strongly associated with myocardial infarction, especially in South Asian populations. Elevated LDL-C and triglycerides with low HDL-C levels contribute to atherosclerosis and plaque rupture. Statins remain the mainstay of therapy, while newer agents like PCSK9 inhibitors and inclisiran provide additional LDL reduction. Each 1 mmol/L decrease in LDL-C lowers heart attack risk by about 22%. However, underdiagnosis, poor adherence, and limited access to advanced therapies continue to hinder effective management, highlighting the need for improved screening and ethnicity-based treatment approaches. **Conclusion:** Finally, dyslipidemia is an important and extremely common determinant of myocardial infarction whose global burden is driven by both genetic and lifestyle factors. As extensive research has been conducted on dyslipidemia, there are still significant gaps in early diagnosis, ethnicity stratified risk prediction, and access to sophisticated treatment. This review thus seeks to bring together existing knowledge regarding the epidemiology, pathophysiology, clinical significance, and therapeutic approach of dyslipidemia as well as highlighting areas for further study and intervention.

KEYWORDS: Dyslipidemia, Cardiovascular Disease, Myocardial Infarction, Atherosclerosis, Lipid Abnormalities, Statins, PCSK9 Inhibitors, Heart Attack Prevention.

INTROCUTION

Dyslipidemia is among the largest contributors to cardiovascular disease (CVD) which still remains the principal cause of death and disease globally.[1] Dyslipidemia is a situation where there is an excess or deficiency of lipids in the blood. This is typically indicated by elevated levels of low-density lipoprotein cholesterol (LDL - C), total cholesterol, triglycerides, and/or decreased levels of high-density lipoprotein cholesterol (HDL - C).[2] These lipid issues accelerate the onset of atherosclerosis by saucing endothelial dysfunction, plaque initiation, and ultimately plaque rupture. This can result in a heart attack or stroke.[3] Dyslipidemia has become more prevalent worldwide over the past few decades due to changes in lifestyle, including not exercising sufficiently, poor diet, and increasing numbers of individuals becoming obese and diabetic. It has emerged as a leading cause of premature coronary artery disease in South Asia, particularly India, where it strikes in younger individuals in contrast to Western nations. Familial and hereditary factors render the clinical scenario even more complex, leading to cardiovascular events occurring prematurely.[4] Dyslipidemia is clinically relevant, both because it is closely associated with the risk of myocardial infarction, and because it can be modified. Dyslipidemia may be managed through lifestyle changes, drug therapy, and emerging therapeutic drugs, unlike other non-modifiable risk factors such as age or genetic predisposition.[5] It is extremely crucial to diagnose and treat atherosclerosis disease early enough in an attempt to retard its progression and prevent adverse cardiovascular events.

This review aims to discuss the epidemiology, etiology, and clinical relevance of dyslipidemia in myocardial infarction. It also examines emerging and new treatments, identifies diagnostic issues, and discovers areas of research deficiency that must be addressed in order to improve long - term cardiovascular health outcomes.[6] Also seeks to investigate the epidemiology, pathophysiology, and clinical significance of dyslipidemia in the context of myocardial infarction. It also assesses existing and new therapeutic interventions, underscores diagnostic hurdles, and formulates research gaps that should be addressed to enhance long - term cardiovascular disease outcomes.

LITERATURE REVIEW

Dyslipidemia has been widely studied as one of the major modifiable risk factors of cardiovascular disease and a significant body of research has been conducted on the prevalence, pathophysiology, clinical implications, and treatment approaches. Dyslipidemia is prevalent in 30-40% of the adult population in the world, with a significant increase in the prevalence in South Asia. Sharma et al. 2023 discovered that dyslipidemia occurs significantly more often in women of a heart attack with postmenopausal. According to the results of Singh et at. (2021), LDL-C and HDL-C were the most effective indicators of a heart attack at the young age of adults. These findings are in line with other meta- analyses, such as Silverman et at. (2016) which reported that one mmol/L decrease in LDL0C is linked with a 22% reduction in the risk of myocardial infarction,[7] as well as other studies. Pathophysiological basis of dyslipidemia and the cardiovascular outcome of this pathology has been studied widely. Zhao Newby (2021) explained the molecular pathways of the oxidation of LDL, the formation of foam cells, and plaque rupture, which demonstrated the mechanistic relationship between lipid dysregulation and atherosclerosis.[8] These concepts have given rise to treatment plans which concentrate on LDL-C and other lipoproteins. Lipid profiling and inclusion of apolipoprotein B and lipoprotein measures have been emphasized by both the European Society of Cardiology (ESC) and the American Heart Association (AHA) in relation to high risk patients as far as diagnosis and risk assessment are concerned. As Awan et al. (2024)[9] noted, many patients, particularly in South Asia, are still not properly screened and treated which reflects a significant shortfall in terms of the public health. Statins as the gold standard of lipid management in its therapeutic interventions have always been associated with strong evidence based on the landmark trials such as 4S, HPS, and PROVE-IT TIMI 22. In newer studies, including FOURIER AND ODYSSEY OUTCOMES, PCSH9 inhibitors have demonstrated that by using them, more heart complications can be prevented in high risk individuals. Inclisiran and bempedoic acid are new treatments that appear to lower the lipids but we await long 0 term outcomes data. Durgun and Durmaz (2024)[11] emphasized the growing relevance of combination therapy, and Kallistratos et al. (2024)[12] confirmed the reduction in the risk of recurring myocardial infarction with the use of ezetimibe or PCSK9 inhibitors in statin-based therapies. Finally, research has revealed that gaps in research still exist in dyslipidemia. No risk algorithms consideration of ethnicity is available, particularly among the South Asian population with earlier and more severe coronary artery disease. There is still no long term data on safety and cost efficacy of innovative medicines and the potential of digital health interventions to monitor treatment compliance is yet to be fulfilled. Overall, the literature demonstrates the significance of the treatment and prevention of dyslipidemia as a risk factor of heart disease. Although new drugs have transformed the manner in which we treat individuals, there remains much to be performed in terms of enhancing the identification, correcting faults in treatment, and creating cost-effective and personalized strategies among various categories of individuals.

RESULTS AND DISCUSSION

1. Epidemiology and Frequency

Dyslipidemia is a prevalent metabolic condition worldwide, impacting roughly 40% of individuals. it is very common in South Asia, where it leads to early coronary artery disease, research in India indicates a substantial correlation between dyslipidemia and early

myocardial infarction, particularly affecting young individuals and postmenopausal women.[13] The growing prevalence of obesity, sedentary behavior, and diabetes has exacerbated the occurence.

2. Pathophysiology

Influenced by lipoprotein irregularities, characterized by increased low- density lipoprotein chloreserol (LDL-C), triglycerides (TG), lipoportein (Lp(a)), and decreased high density lipoprotein chloresterol (HDL-C).[14]

Table of Evidence: Dyslipidemia & Heart Attack.

STUDY/SOURCE	YEAR	POPULATION	MAIN FINDINGS
Zhao & Newby (PMC)	2021	Review of molecular studies	LDL oxidation- inflation- plague- rupture
Sharma et al. (India)	2023	120 postmenopausal women	Dyslipidemia significantly more common in mi patients
Singh et al. (Mayo Clinic)	2021	Young adults with premature mi	High LDL, low HDL were strongest predictors of early mi
Koskinas et al. (JAHA)	2020	ACS patients, ECS guidlines	Lipid management crucial in acs; LDL <55 mg/dl recommended
Awan et al. (Pakistan)	2024	300 post- MI patients	Many patients under-tested and undertreated for dyslipidemia
Durgun & Durmaz (MDPI)	2024	Narrative review	Statins+ 1st line, PCSK9 and bempedoic acid emerging
Kallistratos et al. (Canada)	2024	ACS patients	Statins+ ezetimibe+ PCSK9 reduce recurrent mi risk
Anderson et al. (Canada)	2024	National population data	LDL- C & apoB directly linked to mi and CVD
Toth & Branch	2025	ACS survivors	Implementing dyslipidemia guidelines reduces events
Ding et al. (China)	2021	1,200 MI patients with dyslipidemia	High healthcare costs in untreated dyslipidemia
Silverman et al. (JAMA Meta-analysis)	2016	312,000 patients (49 trials)	Every 1 mmol/L LDL-C reduction+~ 22% lower MI risk

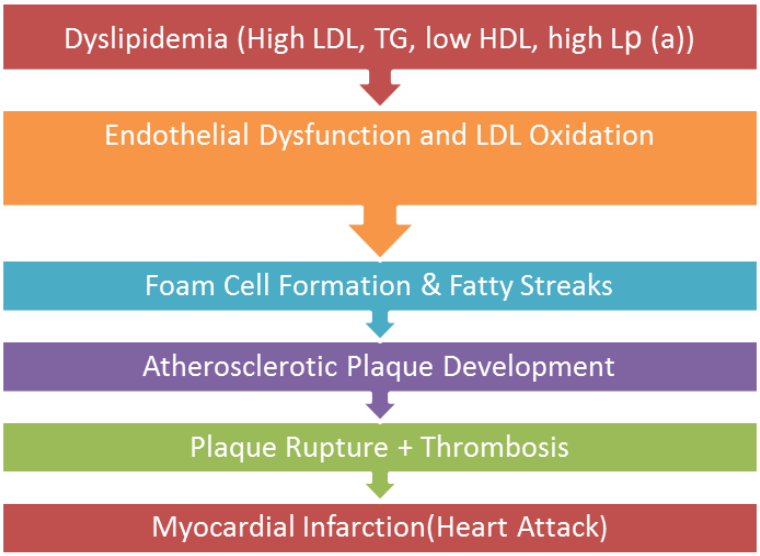

Figure 1: Pathophysiology of Dyslipidemia leading to Myocardial Infraction.

a. Dyslipidemia (LDL, TG, HDL, Lp (a)):[15]
b. This is the initiating risk factor.
c. Endothelial Dysfunction and LDL Oxidation:
d. Excess LDL infiltrates the endothelium and undergoes oxidation. Endothelial dysfunction makes the vessel wall more permeable and promotes inflammation.
e. Foam Cell Formation & Fatty Streaks:
f. Macrophages engulf oxidized LDL- foam cells accumulate- fatty streaks (earliest visible lesion of atherosclerosis).
g. Atherosclerotic Plaque Development:
h. Smooth muscle cell proliferation+ collagen deposition+ lipid core form a fibrous plaque.
i. Plaque Rupture + Thrombosis:
j. Vulnerable plaques rupture - exposure of lipid core and tissue factor- platelet aggregation + thrombus formation.
k. Myocardial Infarction (Heart Attack):
l. Thrombus occludes coronary artery - ischemia and infarction of myocardium.
m. Endothelial Dysfunction and LDL Oxidation: Too much LDL gets into the vascular endothelium, where it oxidizes and starts and inflammatory reaction.
n. Foam cell formation and fatty streaks: Macrophages eat oxidized LDL to make foam cells, which build up as fatty streaks. These are the visual signs of atherosclerosis.
o. Plaque Development and Rupture: Smooth muscle proliferation and lipid core accumulation over time create fibrous plaques.
p. This cascade illustrates the significance of dyslipidemia as a controllable yet pivotal risk factor in cardiovascular events.

3. Clinical manifestations and diagnosis

Dyslipidemia is typically asymptomatic, underscoring the importance of early identification. clinical signs are apparent solely upon the onset of atherosclerosis and ischemic consequences, such angina, transient ischemic episodes, or myocardial infarction. testing a person's lipid profiles is usually how doctors figure out what's wrong with them. chloresterol level LDL HDL-C Triglycerides Apoliporprotein B (apoB) and Lp(a) levels in people who are at high risk.[16]

The European society of cardiology (ESC) and the American Heart Association (AHA) both say that anyone with diabetes, obesity, high blood pressure, or a family history of early CVD should get regular screenings.[17]

4. Therapeutic interventions

To treat dyslipidemia, you can make adjustments to your lifestyle, use medications, and try new treatments:

Lifestyle changes

The initial steps are to eat a diet low in saturated fat and high in fiber, lose weight, quit smoking, and exercise regulary. a weight decrease of just 5-10% can make a big difference in lipid levels and insulin sensitivity.[18]

Pharmacological Treatment

Statins are still the best option; they can cut LD-C by upto 55% and lower the risk of heart disease by aobut 30%. When used alongside statins, ezetimibe (a cholesterol absorption inhibitor) works even better.[19] PCSK9 inhibitors (such evolocumab and alirocumab) and inclisiran lower LDL levels a lot and they work especially well for familial hypercholesterolemia.[8] Bempedoic acid and fibrates are becoming more popular as add-on treatments, especially for people with excessive triglycerides. Digital healtlh tools, yoga, and nutraceuticals are being looked into as ways to increase adherence and overall wellness.[20]

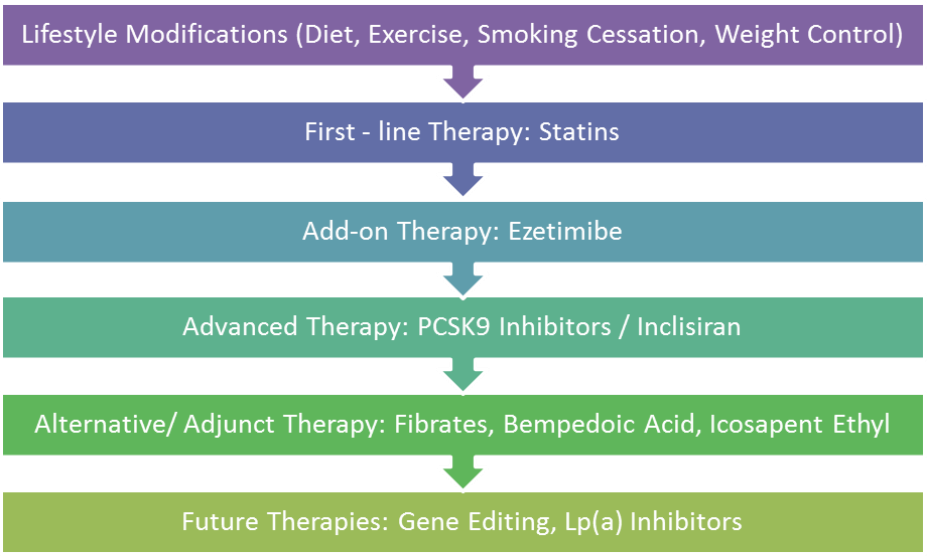

Figure 2: Therapeutic Interventions for Dyslipidemia in MI Prevention.

Table: Lipid - Lowering Therapies with Exact % Effects.

Drug/Class	Mechanism	Typical Lipid Effect (% change)	Key outcome Evidence	Side Effects/ Notes
Statins (Atorvastatin 80 mg, Rosuvastatin 40 mg, Simvastatin 40 mg)	Inhibit HMG- CoA reductase- LDL receptor activity	LDL 50-55% (High-intensity: atrovastatin 80 mg, rosuvastatin 40 mg); LDL 30-49% (moderate intensity: simvastatin 40mg, atorvastatin 10- 20 mg, atorvastatin 10-20 mg); TG 10-30%; HDL 5-10%	4S, HPS, PROVE-IT TIMI 22: Statins MI & CV death by ~ 30%	Myalgias, mild LFTs, rare rhabdomyolysis, first- line, cheap, lifelong.
Ezetimibe (10 mg daily)	Inhibits intestinal cholesterol absorption (NPC1L1	LDL 18-25%	Improve- IT: Ezetimibe+ simvastatin recurrent MI & stroke	Very safe; add-on when statin insufficient
PCSK9 inhibitors (Evolocumb 140 mg Q2W, Alirocumab 75-150 mg Q2W	Prevent PCSK9 from degrading LDL receptors	LDL 50-65% on top of statins	FOURIER (evolocumab), ODYSSEY OUTCOMES (alirocumab): MI, Stroke, CV death	Injection- site reactions, cost high. Excellent in FH & High-risk.
Inclisiran (284mg SC, day 0, 3 months, then q6m)	SiRNA- inhibits hepatic PCSK9 synthesis	LDL-~ 40-50%	ORION trials: robust LDL lowering; outcomes pending	Twice- yearly injection; good adherence; outcomes awaited.
Bempedoic acid (180 mg daily)	Inhibits ATP citrate lyase (upstream of HMG-CoA reductase)	LDL 17-28%	CLEAR Outcome: MACE in statin-intolerant	Can uric acid, gout, tendon rupture (rare)
Fibrates (Fenofibrate 145mg, Gemfibrozil 600 mg BID)	PPAR agonist- lipolysis of TG-RICH LIPOPROTEING	TG 30-50% HDL 5-20%; LDL variable	ACCORD Lipid: neutral overall, benefit in high TG/ low HDL	Aviod gemfibrozil with statins (myopathy risk).

Problems and results untreated or inadequately managed dyslipidemia leads to significant complications

Cardiovascular: Myocardial infarction, angina, stroke, and peripheral arterial disease.

Metabolic: Diabetes mellitus and metabolic syndrome.

Economic Burden: High healthcare expenses because t hospital stays and long term treatment. Large meta-analyses reveal that lowering LDL-C by 1 mmol/L lowers the risk of myocardial infarction by about 22%. This shows how important it is to act quickly.

Research Gaps and Future Directions

Even though things have improved, there are still some gaps: there are not a lot of risk assessment systems that are applicable for particular ethnic groups, particularly South Asian groups. What is still lacked is information about the long term effects of newer anti-lipid lowering drugs like inclisiran and bempedoic acid. Genetic screening need of familial hypercholesterolemia. digital health tools are not being used, as much as it is required to follow up on adherence.

FINDINGS

Literature review and available clinical evidence show that dyslipidemia is invariably linked with an elevated risk of myocardial infarction in various populations. Notable findings are:

Epidemiology: Dyslipidemia exists in 30 to 40% of adult populations worldwide with relatively high prevalence disproportionately concentrated in South Asia, where premature myocardial infarction is prevalent.

Pathophysiology: High LDL- C and triglycerides, along with low HDL - C, lead to endothelial dysfunction, LDL oxidation, foam cell development, and atherosclerotic plaque formation.

Clinical Features: Dyslipidemia is often asymptomatic until an acute coronary event happens, emphasizing the need for early screening and lipid profiling.

Management: Lifestyle changes continue to be the cornerstone of therapy, supplemented by pharmacological treatments such as statins, ezetimibe, PCSK9 inhibitors, and novel agents such as inclisiran.

Complications: Dyslipidemia if left untreated results not only in myocardial infarction but also in increased risk of stroke, peripheral arterial disease, and chronic cardiovascular morbidity.

Research Gaps: Ongoing challenges are under-diagnosis in the developing world, inadequate adherence to recommendations, absence of ethnicity-based algorithms, and sparse long-term outcome data for new agents.

In short, the evidence reaffirms that dyslipidemia is a preventable and treatable condition, but its optimization of management is dependent on both clinical innovation and public health approaches.

CONCLUSION

Dyslipidemia is an important, but extremely modifiable risk factor on the development of atherosclerotic cardiovascular disease, such as a myocardial infarction. Its increasing global prevalence, especially among South Asian groups, is an indicator of the merging of genetic susceptibility and rapidly changing lifestyle patterns, which are marked by unhealthy diets, a lack of physical activity and increasing rates of obesity and diabetes. The combination of environmental and genetic factors makes dyslipidemia a major problem in the medical and general health field. The evidence speaks volumes: Lipid issues exacerbate atherosclerosis by endothelial dysfunction, LDL oxidation, foam cell formation, plaque expansion, and rupture are capable of causing both acute heart diseases such as heart attacks and chronic diseases such as stroke, peripheral artery disease, and long term heart impairment. Dyslipidemia may often remain without any symptoms until a critical event occurs and so early diagnosis, periodic screening and vigorous treatment are essential. The treatment of dyslipidemia requires a combined approach. The fundamental basis of therapy is lifestyle change, which includes the changes in diet, weight loss, and exercise, which can significantly improve lipid profiles and reduce the general risk. However, the mere change of lifestyle is not sufficient to some patients. Pharmaceutical treatment options such as statins, ezetimibe, fibrates, PCSK9 inhibitors, and inclisiran are beneficial in particular cases. Not only do such treatments reduce the cholesterol levels, they also significantly reduce the number of heart attacks and deaths. Care is also complex with the introduction of other approaches such as digital health monitoring, nutraceuticals, and personalized medicine. Despite this advancement, a lot of issues remain that should be resolved. Guideline- directed therapy remains not popular particularly in the low and middle income nations whereby the population with dyslipidemia is increasing the fastest rate. There is a failure to establish ethnicity specific risk algorithms, and this may lead to the underestimation of cardiovascular risk in a risk group such as South Asians. Even new lipid lowering drugs are promising but they are too costly and not very common, therefore they cannot fit in the ordinary practice. Research on long term outcomes, genetic screening and the inclusion of psychosocial and economic variables in care plans also have gaps in research. To sum up, dyslipidemia cannot be discussed merely as a laboratory diagnosis but as a serious systematic disease that has far-reaching consequences. In order to prevent and cure diseases, we must have clinical interventions and public health policies that would promote. Healthcare becomes more accessible and increases awareness through healthy living. Research in the future should be directed at identifying methods of management that can be more individualized and affordable and culturally suitable. It is only through such extensive efforts that we can reduce the global burden of dyslipidemia and reduce the appalling impacts of heart attacks and other cardiovascular disease incidents.

Research should be conducted on the concept of personalised medicine in the future, which integrates genetic, lifestyle, and digital health data in order to optimise Dyslipidemia therapeutic intervention and reduce the burden of cardiovascular disease.

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