

“Lipid Abnormalities and Acute Coronary Syndrome”: A Focused Review on Dyslipidaemia as a Major Risk Factor

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Date of Submission: 05-09-2025

Date of Acceptance: 15-09-2025

ABSTRACT

Dyslipidemia is a key modifiable risk factor for acute coronary syndrome (ACS), significantly contributing to the development and progression of coronary artery disease. This review integrates findings from recent studies focusing on the relationship between lipid abnormalities and ACS outcome. The review highlights persistent challenges in implementing lipid lowering guidelines, the residual risk associated with small dense LDL particles, and the emerging significance of lipid related indices such as the triglyceride-glucose index and triglyceride / HDL-C ratio. The findings emphasize the need for a more personalized and proactive approach in managing dyslipidemia to reduce recurrent cardiovascular events in ACS patients. Strengthening clinical adherence to guidelines and improving early screening can greatly enhance cardiovascular outcomes.

KEY WORDS: Acute coronary syndrome, Lipid abnormalities, Dyslipidemia

I. INTRODUCTION

Dyslipidemia, an abnormal level of lipids in the blood, is a major risk factor for acute coronary syndrome (ACS). ACS includes conditions like heart attacks and unstable angina, where blood flow to the heart is suddenly blocked. Managing lipid levels effectively is key to preventing and treating ACS. Recent studies have looked at different types of lipids, treatment strategies, and risk prediction models to improve patient outcomes.

Etiological factors of acute coronary syndrome

Non-modifiable risk factors

- **Age:** Men are more predominant than women. It is due to women make less estrogen (a female hormone) after menopause. Also, changes in the small blood vessels of the heart when age raises the risk for coronary micro vascular disease.

- **Sex:** Men are more susceptible to heart attacks up until the age of about 55. After menopause, women have equal risks as men. Plus, men tend to have problems in the large arteries of the heart while women often have blockages of smaller arteries in the heart.
- **Family history of CAD:** Those who are having early heart disease younger than 50 year of age have an increased CAD mortality risk^[13].

Modifiable risk factors: Risk factors that can in principle be prevented, changed, Or controlled^[15]

Modifiable risk factors of atherosclerosis include the following;

- Smoking or other tobacco uses
- Dyslipidemia
- Diabetes mellitus
- Hypertension
- Obesity (abdominal obesity)
- Psychosocial stress
- Sedentary lifestyle and/or lack of exercise
- Metabolic syndrome: Metabolic syndrome doubles the risk of developing heart disease when compared to the people who do not have metabolic syndrome.

Complications of acute coronary syndrome

- **Heart failure:** CAD may result in cardiac failure. Heart failure means that heart is unable to pump enough blood through the body. This may leads to fluid build-up in the lungs, difficulty breathing, and swelling of the legs, liver, or abdomen^[14].
- **Abnormal heartbeat:** An abnormal heartbeat is called an arrhythmia. When a person is at rest, the heart is normally beats around 60 to 80 times per minute in a predictable, steady rhythm and with consistent force^[16].

Arrhythmia leads to,

- Bradycardia, a slow heart rate
 - Tachycardia, a fast heart rate
 - Atrial fibrillation, a chaotic, irregular rhythm in the top chambers of the heart (atria).
- Chest pain: Reduced blood flow can mean that your heart doesn't receive enough blood, especially when you exert yourself. This is angina pain. Angina can induce chest numbness or sensations of tightness, heaviness, pressure, and aching, burning, squeezing, fullness^[17].
- Heart attack: If the fatty plaque in one of coronaries arteries ruptures, a blood clot can form. This can greatly block and decrease needed blood flow to the heart, causing a heart attack^[18].
- Sudden death: If blood flow to the heart is severely blocked and not restored, it can cause sudden death^[19].
- Related arterial diseases: The process that can causes damage and plaque accumulation in the coronary arteries might spread to other arteries in the body. The carotid arteries in the neck bring blood to the brain. Atherosclerotic plaques in these arteries can lead to ischemic strokes. Plaques elsewhere can impede blood flow to the legs, arms, or other vital organs, and accumulation of these plaques can lead to aneurysm formation with life-threatening rupture such as aneurysm and rupture of the abdominal aorta or a cerebral artery^[20].

KEY FINDINGS FROM RECENT RESEARCH

A number of recent studies have explored various aspects of dyslipidemia in relation to ACS:

Recent evidence strongly supports the role of dyslipidemia as a central factor in the development, severity, and recurrence of acute coronary syndrome (ACS). Abnormal lipid profiles, including elevated LDL, low HDL and high triglycerides, are consistently linked to more severe coronary artery involvement and poorer clinical outcomes^[3]. Advanced markers such as small dense LDL, non- HDL cholesterol and the triglycerides- glucose index have emerged as more precise indicators of cardiovascular risk, even when traditional lipid targets are met^[4]. These markers also highlight residual risk in patients already undergoing standard lipid-lowering therapy^[2]. Moreover early and aggressive use of combination lipid- lowering treatment is shown to be more

effective in preventing future cardiac events than monotherapy alone^[5]. The TG / HDL-C ratio and related indices are now recognised as powerful predictors of both metabolic and cardiac complications, particularly in patients with stable angina and in a population with high insulin resistance^[10]. Alarming, dyslipidemia is increasingly observed in children and adolescents, indicating the need for early preventive strategies^[11]. Secondary cause of lipid abnormalities, including metabolic and endocrine disorders, also contribute significantly to atherosclerosis and must be addressed during treatment^[12]. Overall, the integrated evidence underscores the importance of a comprehensive, personalized, and proactive approach in diagnosing and managing dyslipidemia to improve long term cardiovascular outcomes in ACS patients.

CLINICAL IMPLICATION OF DYSLIPIDEMIA IN ACS MANAGEMENT

Dyslipidemia continues to be a major therapeutic target in patients with acute coronary syndrome (ACS), given its direct influence on plaque formation, progression and rupture^[3]. The presence of abnormal lipid levels not only increases the likelihood of an initial cardiovascular event but also significantly raises the risk of recurrent episodes^[1]. Current clinical practice guidelines emphasize the importance of achieving strict lipid control, particularly targeting LDL cholesterol below 55 mg/dl in very high risk patients^[5]. However real world challenges such as medication non adherence, therapeutic inertia, and underutilization of combination therapy hinder optimal lipid management^[7]. Furthermore many patients experience 'residual risk' despite reaching LDL goals, underscoring the need to assess other lipid fraction such as non HDL cholesterol, lipoprotein (a), and the triglyceride glucose index^[2]. Integrating these parameters into clinical assessments may allow better risk stratification and personalization of therapy^[6]. The early initiation of high intensity statins, along with the addition of ezetimibe or PCSK9 inhibitors when needed, forms the cornerstones of contemporary ACS management strategies^[9]

FUTURE PERSPECTIVES AND PREVENTIVE STRATEGIES

As cardiovascular disease remains the leading cause of death globally, addressing dyslipidemia early (even before clinical symptoms appear) is crucial^[11]. Emerging research suggests that screening for lipid disorders in adolescence

and early adulthood can significantly delay or prevent the onset of atherosclerotic changes^[4]. In addition, genetic testing and advanced lipid profiling are helping to identify individuals with familial or secondary dyslipidemia, enabling earlier intervention^[12]. Life style modifications, including dietary changes, regular physical activity and smoking cessation, remain foundational to lipid control^[5]. Looking ahead, the future of dyslipidemia management lies in precision medicine – tailoring management not only to lipid level but also to genetic, metabolic, and inflammatory profiles^[6]. Encouraging better patient education and healthcare provider training will also be key to closing the gap between evidence and practice in dyslipidemia care^[8]

II. CONCLUSION

These studies highlight the ongoing challenges of managing dyslipidemia in patients with acute coronary syndrome. While newer treatments and better diagnostic tools (like lipid ratios and glucose indexes) show promise, many patient remains at high risk due to poor guideline implementation or hidden lipid abnormalities. To improve outcomes, healthcare systems must focus on early detection, personalized treatment, and consistent follow-up care. Better education for both patients and providers is essential to address this crucial aspect of heart health.

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