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## LIPOPROTEIN METABOLISM AND ATHEROSCLEROSIS: CLINICAL FEATURES AND LABORATORY DIAGNOSIS

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**Abstract** Atherosclerosis is a chronic inflammatory disease of the arterial wall characterized by lipid accumulation, endothelial dysfunction, and progressive plaque formation. Dysregulation of lipoprotein metabolism, including elevated low-density lipoprotein (LDL) and reduced high-density lipoprotein (HDL) levels, is a key pathogenic factor. Accurate laboratory assessment of lipid profiles and advanced lipoprotein testing is essential for risk stratification, diagnosis, and monitoring of therapeutic interventions. This review summarizes lipoprotein metabolism, the pathophysiology of atherosclerosis, clinical manifestations, and contemporary laboratory diagnostic approaches, in accordance with standards of leading American medical journals.

**Keywords** Lipoproteins; atherosclerosis; lipid metabolism; LDL; HDL; laboratory diagnostics; cardiovascular risk

### Introduction

Atherosclerosis is the leading cause of cardiovascular morbidity and mortality worldwide. The disease is initiated by endothelial injury and modified lipid accumulation within the arterial intima, leading to plaque formation and potential vascular occlusion. Lipoproteins, including chylomicrons, very low-density lipoproteins (VLDL), LDL, and HDL, play a central role in lipid transport and homeostasis. Dyslipidemia, particularly elevated LDL cholesterol and triglycerides, and low HDL cholesterol, is strongly associated with atherosclerotic cardiovascular disease (ASCVD).[1,2,3]

Timely identification of dyslipidemia through laboratory diagnostics is crucial for risk assessment, early intervention, and prevention of cardiovascular events. Recent advances in lipoprotein subfraction analysis and apolipoprotein measurements provide more precise evaluation of cardiovascular risk.

### Lipoprotein Metabolism

Lipoproteins are complexes of lipids and proteins that transport hydrophobic molecules in plasma.

**Chylomicrons:** Transport dietary triglycerides and cholesterol from the intestine to peripheral tissues.

**VLDL:** Produced by the liver, carry endogenous triglycerides.

**LDL:** Formed from VLDL metabolism; primary carrier of cholesterol to peripheral tissues. High LDL levels contribute to plaque formation.

**HDL:** Mediates reverse cholesterol transport from tissues to the liver, providing atheroprotective effects.[4]



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Dysfunction in these pathways, whether genetic (e.g., familial hypercholesterolemia) or acquired (e.g., metabolic syndrome), accelerates atherogenesis.

#### Pathophysiology of Atherosclerosis

Atherosclerosis is a multifactorial process involving:

1. Endothelial Dysfunction: Reduced nitric oxide bioavailability, increased permeability.
2. Lipoprotein Retention and Oxidation: LDL particles accumulate in the intima and undergo oxidative modification.
3. Inflammatory Response: Recruitment of monocytes/macrophages leads to foam cell formation.
4. Plaque Progression: Smooth muscle cell proliferation, extracellular matrix deposition, and calcification occur.
5. Complications: Plaque rupture may result in thrombosis, myocardial infarction, or stroke.

#### Clinical Features

Often asymptomatic until advanced disease.[5]

Stable Angina: Chest pain with exertion due to partial coronary occlusion.

Acute Coronary Syndromes: Myocardial infarction or unstable angina due to plaque rupture.

Peripheral Artery Disease: Claudication and impaired perfusion in lower extremities.

Cerebrovascular Events: Stroke or transient ischemic attacks.

#### Laboratory Diagnostics

##### 1. Conventional Lipid Profile

Total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides.

Friedewald formula commonly used to estimate LDL.

##### 2. Advanced Lipoprotein Testing

Apolipoprotein B (ApoB): Marker of atherogenic particle number.

Apolipoprotein A-I (ApoA-I): Principal protein in HDL; inversely related to risk.

Lipoprotein(a) [Lp(a)]: Independent cardiovascular risk factor; genetically determined.

LDL Particle Size and Number: Assessed via nuclear magnetic resonance (NMR) spectroscopy or ultracentrifugation.

##### 3. Biomarkers of Inflammation

High-sensitivity C-reactive protein (hsCRP): Indicates vascular inflammation and predicts ASCVD events.

Interleukins and adhesion molecules: Research-level markers for endothelial activation.

##### 4. Genetic Testing

Familial hypercholesterolemia: Mutations in LDLR, APOB, or PCSK9 genes.

Prevention and Management



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Lifestyle Modification: Diet (low saturated fat, increased fiber), exercise, smoking cessation.

Pharmacotherapy: Statins, ezetimibe, PCSK9 inhibitors, fibrates, or niacin depending on lipid profile.

Monitoring: Regular laboratory assessment of lipid levels, apolipoproteins, and inflammatory markers.

Population Screening: Early detection in high-risk individuals to prevent cardiovascular events.[6,7,8]

### **Conclusion**

Dysregulation of lipoprotein metabolism is central to the development of atherosclerosis. Laboratory diagnostics, including conventional lipid profiles, advanced lipoprotein testing, inflammatory biomarkers, and genetic analysis, are critical for risk stratification, early detection, and monitoring of therapeutic interventions. Integrating clinical evaluation with precise laboratory data enhances cardiovascular prevention and management strategies.

### **REFERENCES:**

- 1.Grundy SM, Stone NJ, et al. 2018 AHA/ACC Guideline on the Management of Blood Cholesterol. *Circulation*. 2019;139:e1082–e1143.
- 2.Ference BA, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease. *J Am Coll Cardiol*. 2017;70:66–77.
- 3.Catapano AL, et al. ESC/EAS Guidelines for the management of dyslipidaemias. *Eur Heart J*. 2016;37:2999–3058.
- 4.Toth PP. Apolipoprotein measurements in clinical practice. *J Clin Lipidol*. 2016;10:24–33.
- 5.Raal FJ, Santos RD. Homozygous familial hypercholesterolemia: current perspectives. *Curr Opin Lipidol*. 2012;23:233–240.
- 6.Makhamatov U. Anemia Disease and Rational Nutrition in it. – 2023.
- 7.Maxamatov U., To‘lkinov I., Xabibullayeva M. Eating Habits in Hematological Diseases. – 2023.
- 8.Maxamatov U. S. Treatment of Triggerral Helmintosis in Children and Adolescents Using Folk Medicine. – 2023

