

LIPID METABOLISM DISORDERS AS A KEY FACTOR IN THE DEVELOPMENT OF ATHEROSCLEROSIS

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Abstract

This article examines the role of lipid metabolism disorders in the development of atherosclerosis. It describes the functions of cholesterol and lipoproteins, the mechanisms of atherosclerotic plaque formation, and the major risk factors associated with dyslipidemia. Particular attention is paid to diagnostic methods, cardiovascular complications, and modern approaches to prevention and treatment. The importance of early detection of lipid abnormalities and comprehensive cardiovascular risk management is emphasized.

Keywords: Atherosclerosis, lipid metabolism, cholesterol, low-density lipoproteins, high-density lipoproteins, dyslipidemia, cardiovascular disease.

Introduction

Atherosclerosis is a chronic disease characterized by lipid accumulation, inflammation, and structural changes in the arterial walls. It is a major underlying cause of coronary heart disease, myocardial infarction, ischemic stroke, and peripheral arterial disease. These conditions contribute significantly to morbidity and mortality worldwide. Lipid metabolism disorders play a central role in atherosclerosis. Elevated concentrations of atherogenic lipoproteins promote cholesterol accumulation in arterial walls and stimulate inflammatory processes. The development of the disease is also influenced by hereditary predisposition,

hypertension, diabetes mellitus, smoking, obesity, physical inactivity, and dietary habits.

The purpose of this article is to examine the mechanisms through which lipid metabolism disorders contribute to atherosclerosis and to summarize the principal methods of diagnosis, prevention, and treatment. Cholesterol is essential for normal physiological function. It is a structural component of cell membranes and participates in the synthesis of steroid hormones and bile acids. The body obtains cholesterol from food and produces it primarily in the liver. Because cholesterol and triglycerides are poorly soluble in blood, they are transported by lipoproteins. These particles differ in composition, density, and biological function. Low-density lipoproteins (LDLs) transport cholesterol to peripheral tissues. However, when their concentration remains elevated, LDL particles can enter and become retained in the arterial intima. This process initiates a series of cellular and inflammatory reactions that contribute to atherosclerotic plaque development. The risk of cardiovascular disease depends on both the concentration of atherogenic lipoproteins and the duration of exposure to elevated levels [1-10]. High-density lipoproteins (HDLs) participate in reverse cholesterol transport, carrying cholesterol from peripheral tissues toward the liver for processing and elimination. Although low HDL cholesterol is associated with increased cardiovascular risk, HDL levels should not be interpreted in isolation. Very-low-density lipoproteins (VLDLs), produced mainly in the liver, transport triglycerides to peripheral tissues. Their metabolic products can also contribute to atherosclerosis. Elevated triglyceride levels frequently occur in obesity, insulin resistance, and type 2 diabetes mellitus. Therefore, the assessment of lipid metabolism generally includes total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides. Additional tests, such as apolipoprotein B and lipoprotein(a), may be appropriate in selected patients.

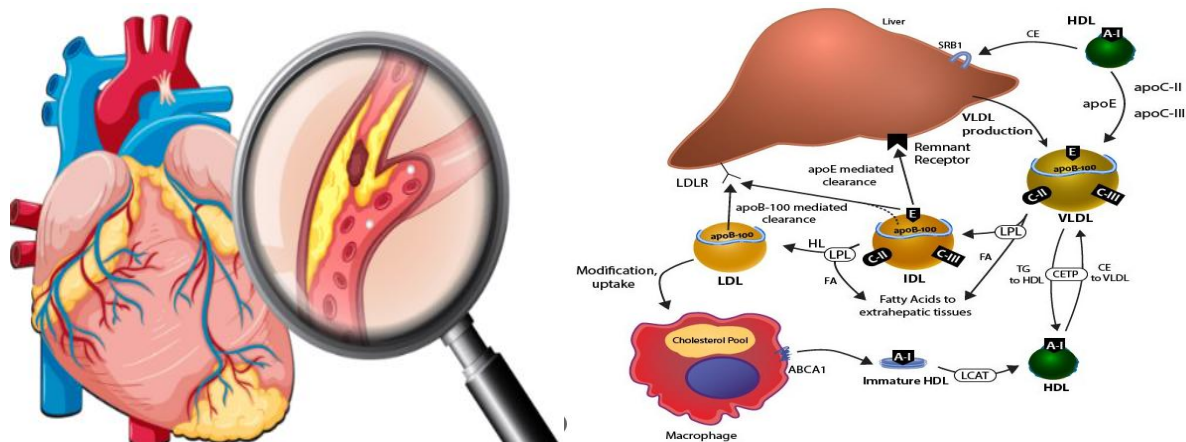


Fig 1. Metabolism of ApoB100 containing lipoproteins.

Atherosclerosis develops through a complex interaction of lipid accumulation, endothelial dysfunction, inflammation, and structural remodeling of the arterial wall.

The vascular endothelium normally regulates vascular tone, blood coagulation, and inflammatory responses. Factors such as smoking, hypertension, and metabolic disorders can impair endothelial function and contribute to an unfavorable vascular environment. A key stage in atherosclerosis is the retention of atherogenic lipoproteins within the arterial intima. These particles may undergo modifications that promote inflammatory signaling. Monocytes migrate into the vessel wall and differentiate into macrophages, which absorb modified lipoproteins. As lipids accumulate, macrophages become foam cells, forming an early characteristic feature of atherosclerosis.

With disease progression, cell death and lipid accumulation contribute to the formation of a lipid-rich core. Smooth muscle cells and connective tissue form a fibrous cap over this core, resulting in an atherosclerotic plaque.

The clinical consequences depend not only on the degree of arterial narrowing but also on plaque stability. Plaque rupture or erosion can trigger platelet activation and thrombosis, potentially causing acute myocardial infarction or ischemic stroke.

Thus, atherosclerosis is not simply the deposition of fat in blood vessels; it is a chronic inflammatory process in which elevated atherogenic lipoproteins play a central role.

Dyslipidemia refers to abnormal concentrations or composition of lipids and lipoproteins in the blood. It may be primary, resulting from hereditary factors, or secondary, developing because of other diseases, medications, or lifestyle factors. Familial hypercholesterolemia is an important inherited disorder characterized by elevated LDL cholesterol from an early age. Prolonged exposure to high LDL levels increases the likelihood of premature atherosclerotic cardiovascular disease. A family history of early myocardial infarction or stroke may indicate the need for earlier screening and evaluation of relatives. Secondary dyslipidemia may be associated with diabetes mellitus, obesity, insulin resistance, hypothyroidism, chronic kidney disease, and certain liver disorders. Some medications can also alter lipid levels. Identifying the underlying cause is important for selecting appropriate treatment.

Lifestyle factors significantly influence lipid metabolism. Excessive calorie intake, diets high in saturated fats and added sugars, insufficient physical activity, and excessive alcohol consumption can contribute to unfavorable lipid profiles. Smoking additionally damages the endothelium and increases cardiovascular risk. The combined effects of several risk factors are particularly important. For example, dyslipidemia accompanied by hypertension and diabetes may substantially increase the likelihood of cardiovascular complications. Consequently, risk assessment should consider the patient's overall clinical condition rather than cholesterol levels alone.

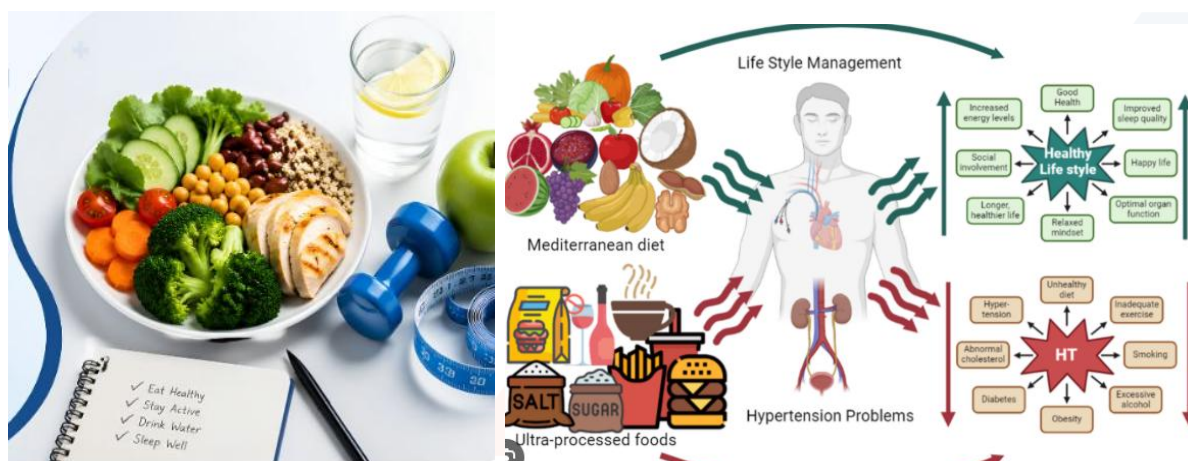


Fig.2 The interactions between aging and metabolic diseases.

Dyslipidemia is often asymptomatic, making laboratory screening essential for identifying individuals at increased cardiovascular risk. Diagnosis begins with a medical history, assessment of family history and lifestyle, measurement of blood pressure, and evaluation of relevant chronic diseases. The lipid profile is the primary laboratory investigation. Depending on the clinical situation, additional tests may include blood glucose, glycated hemoglobin, thyroid function, and kidney and liver function assessments. Apolipoprotein B and lipoprotein(a) measurements can provide further information about atherogenic risk in selected individuals [5-14].

Lipid results should be interpreted according to overall cardiovascular risk. Patients with established cardiovascular disease generally require more intensive risk reduction than individuals without diagnosed disease. Treatment targets are determined using current clinical guidelines and individual patient characteristics. Lifestyle modification is fundamental to prevention. A balanced diet emphasizing vegetables, fruits, legumes, whole grains, nuts, and unsaturated fats can improve cardiovascular health. Replacing saturated fats with unsaturated fats, maintaining a healthy body weight, engaging in regular physical activity, and avoiding tobacco are important preventive measures. Limiting excessive alcohol consumption and controlling blood pressure and blood glucose are also beneficial.

When lifestyle changes are insufficient or cardiovascular risk is high, pharmacological treatment may be necessary. Statins are commonly used to lower LDL cholesterol and reduce the risk of cardiovascular events. Depending on the patient's condition and treatment response, additional lipid-lowering medications may be prescribed by a healthcare professional. Long-term management requires regular monitoring, adherence to treatment, and control of associated risk factors. Early identification and sustained reduction of elevated atherogenic lipoproteins can help prevent or delay atherosclerotic complications.

Conclusion: Lipid metabolism disorders are among the most important modifiable factors contributing to atherosclerosis. Elevated LDL cholesterol and other atherogenic lipoproteins promote lipid retention, inflammation, and plaque formation in arterial walls. Hereditary predisposition, metabolic diseases, smoking, hypertension, and unhealthy lifestyle habits further increase cardiovascular risk.

Effective prevention requires early diagnosis, comprehensive risk assessment, appropriate dietary and physical activity changes, and lipid-lowering therapy when indicated. An individualized and sustained approach to managing dyslipidemia is essential for reducing cardiovascular complications and improving long-term health.

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