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MORPHOLOGICAL ASSESSMENT OF CORONARY ATHEROSCLEROTIC PLAQUES AND THEIR CLINICAL SIGNIFICANCE

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Abstract.Background: Coronary atherosclerosis is a chronic inflammatory and metabolic disorder characterized by lipid accumulation, endothelial dysfunction, and progressive structural alterations of the arterial wall. The development of atherosclerotic plaques may result in coronary artery narrowing, impaired myocardial perfusion, and potentially life-threatening cardiovascular complications.**Objective:** To review the morphological characteristics, pathological progression, and clinical significance of atherosclerotic plaques in human coronary arteries.**Materials and Methods:** This article presents a narrative review of scientific publications addressing the pathogenesis, histological structure, morphological classification, and clinical consequences of coronary atherosclerosis. Particular attention is given to lipid accumulation, inflammatory cell infiltration, fibrous cap formation, plaque calcification, and mechanisms of plaque instability. **Results:** Coronary atherosclerotic lesions demonstrate considerable morphological variability, ranging from early fatty streaks to advanced fibroatheromas and complicated plaques. Their development involves endothelial injury, retention and modification of lipoproteins, macrophage activation, foam cell formation, and proliferation of vascular smooth muscle cells. Plaques with large lipid-rich necrotic cores, inflammatory infiltration, and thin fibrous caps are particularly susceptible to rupture. Plaque disruption can initiate platelet activation and thrombus formation, leading to acute coronary syndromes.**Conclusion:** Morphological evaluation of coronary atherosclerotic plaques provides important information about disease progression and cardiovascular risk. Understanding the structural characteristics of different plaque phenotypes may contribute to improved diagnostic approaches, risk stratification, and prevention of coronary complications.

Keywords: Coronary atherosclerosis, coronary arteries, endothelial dysfunction, lipid accumulation, inflammation, plaque instability

Introduction

Atherosclerosis represents one of the principal pathological processes underlying cardiovascular disease and remains a major cause of morbidity and mortality worldwide. Coronary artery involvement is particularly important because progressive arterial narrowing and acute thrombotic complications can compromise myocardial blood supply.

The development of coronary atherosclerosis is a multifactorial process involving interactions between circulating lipoproteins, endothelial cells, inflammatory mediators, vascular smooth muscle cells, and components of the extracellular matrix. The accumulation of cholesterol-containing lipoproteins within the arterial intima represents an important initiating event in plaque formation.In the early stages, atherosclerotic lesions may appear as fatty streaks containing lipid-

loaded macrophages. With disease progression, these lesions undergo structural transformation, resulting in the formation of fibrous plaques and lipid-rich necrotic cores. Advanced lesions may become unstable and undergo surface erosion or rupture, promoting intravascular thrombosis.The morphological characteristics of coronary plaques are closely associated with their clinical behavior[1]. While some lesions predominantly produce chronic luminal narrowing, others may remain clinically silent until acute plaque disruption occurs. Therefore, understanding the histopathological features of coronary atherosclerosis is essential for evaluating its progression and complications.

Materials and Methods

This narrative review focuses on published experimental, pathological, and clinical investigations concerning human coronary atherosclerosis. Scientific literature addressing coronary plaque development, histological classification, endothelial dysfunction, lipid metabolism, inflammation, and plaque rupture was considered.The main aspects of analysis included:

Morphological characteristics of early and advanced atherosclerotic lesions.Lipid deposition and formation of foam cells.Inflammatory mechanisms involved in plaque development.

Structural changes in the fibrous cap.Plaque calcification and necrotic core formation.

Mechanisms of plaque rupture, erosion, and coronary thrombosis.The review emphasizes findings from established pathological studies and contemporary cardiovascular research.

Results

Early morphological changes in coronary arteries

The earliest manifestations of coronary atherosclerosis are associated with functional and structural alterations of the vascular endothelium. Increased endothelial permeability and retention of apolipoprotein B-containing lipoproteins within the intimal layer contribute to the initiation of atherosclerotic lesions.

Modified lipoproteins stimulate inflammatory responses and promote the recruitment of circulating monocytes. Following their migration into the arterial intima, monocytes differentiate into macrophages and accumulate intracellular lipids, forming foam cells.

These changes result in the development of fatty streaks, which represent the earliest recognizable morphological stage of atherosclerosis[2].

Formation and structural organization of atherosclerotic plaques

As the disease progresses, inflammatory activity and lipid accumulation contribute to the development of more complex arterial lesions.

The principal structural components of an established atherosclerotic plaque include:

Fibrous cap consisting primarily of collagen and smooth muscle cells.

Lipid-rich necrotic core containing cholesterol crystals and cellular debris.

Inflammatory infiltrates, particularly macrophages and T lymphocytes.

Extracellular matrix components[3].Calcified deposits of varying size and distribution.

The relative proportions of these components determine plaque morphology and may influence its mechanical stability. Morphological types of coronary atherosclerotic plaques

Coronary atherosclerotic lesions demonstrate several morphological patterns.

Fibrous plaques: These lesions are characterized by a relatively dense collagen-rich cap and variable lipid accumulation. They may progressively reduce the arterial lumen.

Fibroatheromas: These plaques contain a prominent lipid-rich necrotic core covered by fibrous tissue. Their composition and inflammatory activity influence their susceptibility to destabilization.Thin-cap fibroatheromas: These lesions possess a large necrotic core and a relatively thin fibrous cap with substantial macrophage infiltration. They are associated with an increased risk

of plaque rupture[4].Calcified plaques: These lesions contain mineral deposits within the arterial wall. Calcification may occur in different morphological patterns and is frequently associated with advanced atherosclerotic disease.

Complicated plaques: These include lesions characterized by surface disruption, intraplaque hemorrhage, ulceration, or superimposed thrombosis.

Plaque instability and coronary thrombosis[5]Plaque instability represents a critical stage in the development of acute coronary complications. Inflammatory macrophages release proteolytic enzymes, including matrix metalloproteinases, which contribute to degradation of the extracellular matrix and weakening of the fibrous cap.

When plaque rupture occurs, thrombogenic components of the necrotic core become exposed to circulating blood. This stimulates platelet adhesion, activation of the coagulation cascade, and thrombus formation[6].Depending on the extent of coronary obstruction, the resulting thrombotic event may cause unstable angina, myocardial infarction, or sudden cardiac death.

Clinical significance of coronary plaque morphology[7]The relationship between plaque structure and clinical manifestations is particularly important in cardiovascular medicine. Stable fibrous plaques are frequently associated with progressive coronary stenosis and exertional angina. In contrast, vulnerable plaques may produce acute coronary events following rupture or erosion.Modern imaging techniques, including coronary computed tomography angiography, intravascular ultrasound, and optical coherence tomography, provide additional opportunities to evaluate plaque composition, calcification, fibrous cap characteristics, and arterial remodeling[8].

Discussion

The morphological progression of coronary atherosclerosis reflects the interaction of lipid metabolism, inflammatory activation, endothelial dysfunction, and vascular remodeling.

Previous pathological investigations have demonstrated that atherosclerotic lesions can develop long before the appearance of clinical symptoms. The PDAY Study particularly emphasized the presence of early atherosclerotic changes in young individuals and demonstrated the importance of conventional cardiovascular risk factors in lesion development.

The transformation of early fatty streaks into advanced fibroatheromas is accompanied by increasing lipid deposition, macrophage accumulation, smooth muscle cell migration, and extracellular matrix remodeling.An important aspect of coronary atherosclerosis is the distinction between plaque burden and plaque vulnerability. Severe luminal narrowing does not necessarily indicate the presence of the most rupture-prone lesion. Some plaques with relatively limited stenosis may nevertheless possess morphological characteristics associated with acute coronary events.These observations support the importance of comprehensive assessment of coronary plaque composition rather than relying exclusively on the degree of arterial stenosis.

Conclusion

Coronary atherosclerosis is a progressive pathological process characterized by lipid accumulation, chronic inflammation, and structural remodeling of the arterial wall. Its morphological manifestations range from early fatty streaks to advanced fibrous and complicated plaques.The composition of atherosclerotic lesions, particularly the size of the necrotic core, thickness of the fibrous cap, inflammatory cell infiltration, and degree of calcification, plays an important role in determining plaque stability.A better understanding of coronary plaque morphology may improve the interpretation of cardiovascular imaging findings, identification of high-risk lesions, and development of preventive and therapeutic strategies aimed at reducing the incidence of acute coronary events.

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