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PATHOLOGICAL ANATOMY OF DIABETIC MACROANGIOPATHY AND ATHEROSCLEROSIS

Karimova Vasila Aslidin kizi

Andijan State Medical Institute, Faculty of Medicine, 3rd-year student

Feruzaxon Sayidjonova

Scientific supervisor: Andijan State Medical Institute

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Abstract

Background:

Diabetes mellitus is a chronic metabolic disorder associated with long-term vascular complications that significantly increase morbidity and mortality. Among these complications, diabetic macroangiopathy plays a central role and is closely linked to accelerated atherosclerosis affecting large and medium-sized arteries.

Objective:

This article aims to describe the pathological anatomical features of diabetic macroangiopathy and atherosclerosis, emphasizing structural changes in the arterial wall and their clinical significance.

Methods:

The study is based on a comprehensive review and analysis of classical and contemporary pathological anatomy literature, focusing on morphological changes observed in major vascular beds in patients with diabetes mellitus.

Results:

Diabetic macroangiopathy is characterized by early onset and rapid progression of atherosclerotic lesions. Pathological findings include endothelial dysfunction, increased lipid permeability of the arterial wall, formation of diffuse and multiple atherosclerotic plaques, medial atrophy, and a high tendency toward thrombosis. These changes predominantly affect the coronary, cerebral, and peripheral arteries, leading to ischemic heart disease, stroke, and peripheral arterial disease, including diabetic foot syndrome.

Diabetic macroangiopathy represents a major pathological and clinical challenge in patients with diabetes mellitus. Understanding its pathological anatomical basis is essential for early diagnosis,

prevention, and effective management of cardiovascular complications. Strict glycemic control and correction of lipid metabolism disorders remain key strategies in reducing disease progression and improving patient outcomes.

Introduction

Diabetes mellitus is currently regarded not only as an endocrine disorder but also as a systemic disease that affects nearly all organs and tissues of the human body. One of the most serious and life-threatening consequences of long-standing diabetes is damage to the cardiovascular system. In the majority of patients with diabetes, the leading cause of morbidity and mortality is cardiovascular disease, which highlights the clinical importance of diabetic macroangiopathy.

Diabetic macroangiopathy is characterized by damage to large and medium-sized arteries and is most often associated with the development of atherosclerosis. In patients with diabetes, atherosclerosis tends to develop at an earlier age, progresses more rapidly, and leads to more severe complications compared to the general population. These changes are primarily driven by chronic hyperglycemia, lipid metabolism disorders, and endothelial dysfunction.

Main Body

In diabetes mellitus, the vascular wall is no longer a passive mechanical structure but becomes an active participant in pathological metabolic processes. Persistently elevated blood glucose levels cause direct injury to endothelial cells, impairing their protective and regulatory functions. As a result, the vascular endothelium becomes more permeable to lipids and pro-atherogenic substances.

From a pathological anatomical perspective, diabetic macroangiopathy represents a complex and progressive process involving structural remodeling of the arterial wall. Chronic metabolic disturbances characteristic of diabetes mellitus lead to sustained endothelial injury, which is considered the earliest and most critical event in the development of macrovascular complications.

Endothelial dysfunction in diabetes is manifested by reduced nitric oxide production, increased expression of adhesion molecules, and enhanced permeability of the vascular wall. These changes facilitate the adhesion and migration of monocytes into the intima, where they differentiate into macrophages and actively uptake modified lipoproteins. As a result, lipid-laden foam cells accumulate and form the initial atherosclerotic lesions.

As the disease progresses, smooth muscle cells migrate from the media into the intima and undergo proliferation. These cells synthesize extracellular matrix components, including collagen and proteoglycans, contributing to the formation of a fibrous cap over the lipid-rich necrotic core. In diabetic patients, this process is markedly accelerated, and the fibrous cap is often thinner and more prone to rupture, increasing the risk of acute vascular events.

Another important pathological feature of diabetic macroangiopathy is medial degeneration. Histological examination frequently reveals thinning and atrophy of the medial layer, fragmentation of elastic fibers, and replacement of normal vascular architecture with fibrotic tissue. These alterations reduce arterial compliance and further compromise blood flow, especially in peripheral arteries.

Inflammation plays a central role in the progression of atherosclerosis in diabetes. Persistent hyperglycemia promotes oxidative stress and the release of pro-inflammatory cytokines, which amplify vascular injury and destabilize atherosclerotic plaques. Plaque instability is a key determinant of thrombosis, particularly in coronary and cerebral arteries, where sudden plaque rupture may lead to myocardial infarction or ischemic stroke.

In the peripheral circulation, diabetic macroangiopathy is often combined with microvascular damage, resulting in severe tissue hypoxia. This dual vascular impairment explains the poor healing capacity observed in diabetic patients and the high incidence of chronic ulcers and gangrene. Pathological examination of affected limbs frequently demonstrates extensive arterial narrowing, occlusive thrombosis, and secondary infectious changes.

Overall, the pathological anatomy of diabetic macroangiopathy reflects a continuous interaction between metabolic dysregulation, vascular inflammation, and structural degeneration of arterial walls. These processes form the morphological basis for the major cardiovascular and peripheral complications associated with diabetes mellitus.

Low-density lipoproteins (LDL) easily penetrate the intima of the arterial wall, where they undergo oxidative modification. This process triggers a chronic inflammatory response involving macrophages and smooth muscle cells, ultimately leading to the formation of atherosclerotic plaques. In diabetic patients, these plaques are typically diffuse, multiple, and symmetrically distributed, affecting several vascular territories simultaneously.

The coronary arteries are particularly vulnerable to diabetic macroangiopathy. Progressive narrowing of the coronary lumen results in myocardial ischemia, which may clinically manifest as stable angina, acute coronary syndrome, or myocardial infarction. Similarly, atherosclerotic involvement of cerebral arteries significantly increases the risk of ischemic stroke.

In the arteries of the lower extremities, diabetic macroangiopathy often leads to chronic ischemia. Reduced blood supply to peripheral tissues results in intermittent claudication, poor wound healing, trophic ulcers, and, in severe cases, tissue necrosis and gangrene. These pathological changes form the anatomical basis of the diabetic foot syndrome, one of the most disabling complications of diabetes.

Another important feature of diabetic macroangiopathy is the increased tendency toward thrombosis. Enhanced platelet aggregation, reduced fibrinolytic activity, and endothelial injury create favorable conditions for thrombus formation on the surface of atherosclerotic plaques. Acute thrombosis may completely occlude the vessel lumen, causing sudden and severe ischemia of vital organs.

Conclusion

In conclusion, diabetic macroangiopathy and atherosclerosis represent some of the most severe and prognostically unfavorable complications of diabetes mellitus. From a pathological anatomical perspective, these conditions are characterized by profound structural alterations of the arterial wall, including endothelial damage, atherosclerotic plaque formation, and thrombosis. These changes underlie the development of ischemic heart disease, stroke, and peripheral arterial disease in diabetic patients. Early detection of vascular damage, strict glycemic control, and effective management of

lipid metabolism disorders play a crucial role in slowing the progression of diabetic macroangiopathy. Understanding the pathological anatomy of these processes is essential for improving preventive strategies and reducing cardiovascular mortality among patients with diabetes.

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