

**MODERN LABORATORY DIAGNOSTICS OF
ENDOTHELIAL DYSFUNCTION IN DIABETES MELLITUS**

Badirdinova G.Z.

Master of degree of the department of Clinical laboratory diagnostics
with the course of clinical laboratory diagnostics of the PGF
Samarkand state medical university

Nabiyeva F.S.

Senior lecturer of the department of Clinical laboratory diagnostics
with the course of clinical laboratory diagnostics of the PGF
Samarkand state medical university

Introduction. Diabetes mellitus is a chronic metabolic disease caused by impaired insulin secretion and its action, leading to disorders of carbohydrate, lipid, and protein metabolism. Endothelial dysfunction is one of its most severe complications and is characterized by structural and functional damage to vascular endothelial cells. This article briefly analyzes the key mechanisms, pathophysiological features, and diagnostic biomarkers of endothelial dysfunction in diabetes mellitus.

Diabetes mellitus is one of the most common endocrine disorders, and its global prevalence continues to increase steadily. According to the World Health Organization (WHO), nearly one tenth of the world's population currently suffers from diabetes mellitus, and the number of affected individuals rises by several million each year. Based on its origin, diabetes mellitus is classified into congenital and acquired forms, and according to clinical characteristics, into type 1 and type 2 diabetes [9].

Type 1 diabetes mellitus develops as a result of autoimmune processes and is characterized by the destruction of insulin-producing β -cells of the pancreas. This leads to absolute insulin deficiency, impaired glucose uptake by

peripheral tissues, and persistent hyperglycemia. The disease occurs predominantly in children and adolescents, with genetic predisposition playing a key role, while environmental factors act as triggers for disease onset [8].

Type 2 diabetes mellitus is characterized by relatively preserved or even increased insulin secretion accompanied by reduced tissue sensitivity to insulin. As a result, glucose metabolism is impaired and blood glucose levels increase. This form of diabetes is closely associated with obesity, metabolic disorders, and an unhealthy lifestyle, and is most commonly observed in middle-aged and elderly individuals [1].

The clinical manifestations of diabetes mellitus develop gradually as a result of prolonged hyperglycemia. The earliest and most characteristic symptoms include increased thirst and frequent urination, which are associated with excessive fluid loss due to glucose excretion in the urine. Impaired glucose uptake by cells leads to energy deficiency, resulting in increased hunger and, particularly in type 1 diabetes, progressive weight loss [11].

Patients commonly complain of general weakness, rapid fatigue, drowsiness, and reduced working capacity. Persistent hyperglycemia may cause blurred vision, dryness of the skin and mucous membranes, pruritus, delayed wound healing, and increased susceptibility to infections. Gastrointestinal manifestations such as an acetone odor on the breath, nausea, and vomiting are characteristic of diabetic ketoacidosis, which is more typical of type 1 diabetes. With long disease duration, chronic complications develop, including diabetic neuropathy, trophic ulcers, diabetic foot syndrome, and an increased risk of gangrene. Early and late complications of diabetes mellitus affect multiple organ systems, significantly impair quality of life, and often lead to long-term disability; in many cases, the disease is diagnosed only after complications have occurred [2].

Laboratory investigations play a crucial role in the early diagnosis of diabetes, as they allow detection of disturbances in glucose metabolism before

clinical symptoms appear. Measurements of blood glucose levels, glycated hemoglobin (HbA1c), and the oral glucose tolerance test are essential tools for identifying early and latent forms of the disease and for monitoring high-risk individuals. In recent years, markers of endothelial dysfunction- such as nitric oxide (NO), E- and P-selectins, endothelin, ICAM-1, VCAM-1, and malondialdehyde (MDA)- have gained importance as indicators of early vascular damage and predictors of micro- and macroangiopathic complications, including retinopathy, nephropathy, and atherosclerosis [6]

The endothelium forms the inner lining of blood vessels and acts as an active regulator between blood flow and the vascular wall. It is a metabolically active structure involved in the regulation of vascular tone, maintenance of hemostatic balance, modulation of inflammatory processes, and participation in metabolic and transport functions. Therefore, the endothelium plays a central role in maintaining vascular health and overall homeostasis [4].

In diabetes mellitus, the most serious consequences are associated with vascular complications, including macroangiopathies such as coronary artery disease, cerebrovascular disorders, and peripheral arterial disease, as well as microangiopathies, including diabetic nephropathy, retinopathy, and neuropathy. Endothelial dysfunction represents a key pathogenic mechanism in these processes, and its early detection is clinically important for preventing or slowing the progression of diabetic angiopathies [10].

Endothelial dysfunction is primarily related to impaired secretory activity of endothelial cells and is mediated by various biologically active factors. Its assessment commonly includes decreased nitric oxide availability, increased endothelin-1 levels, elevated expression of adhesion molecules (ICAM-1 and VCAM-1), selectins, and increased markers of oxidative stress such as malondialdehyde [3,11].

Chronic hyperglycemia promotes excessive generation of reactive oxygen species, leading to oxidative stress and endothelial injury. As a result,

vasodilation is impaired, inflammation is enhanced, vascular elasticity is reduced, and the risk of atherosclerosis increases. Elevated levels of ICAM-1 and VCAM-1 reflect inflammatory activation and endothelial damage and are considered important indicators of endothelial dysfunction [7].

E- and P-selectins are adhesion molecules expressed on the endothelial surface and play an important role in inflammatory processes and leukocyte interaction with the vascular wall. E-selectin is mainly synthesized by endothelial cells in response to inflammatory mediators, whereas P-selectin is rapidly activated on platelets and endothelial cells, enhancing leukocyte adhesion and migration. Elevated levels of these selectins in diabetes and cardiovascular diseases indicate impaired vascular wall function [4].

Nitric oxide produced by the endothelium is a key vasodilatory factor, and its synthesis is mediated by endothelial nitric oxide synthase (eNOS). In diabetes mellitus, dysregulation of eNOS activity leads to reduced nitric oxide availability and increased oxidative stress. Endothelin-1 is a potent vasoconstrictor whose levels rise under conditions such as hyperglycemia and other pathological stimuli and are closely associated with the development of vascular complications [12].

In conclusion, endothelial dysfunction represents one of the major mechanisms underlying the development of angiopathies in diabetes mellitus. Biomarkers such as nitric oxide, endothelin-1, ICAM-1, VCAM-1, E- and P-selectins, and malondialdehyde are important indicators of endothelial status, and their assessment is essential for the early detection of diabetic complications and for evaluating disease severity and treatment effectiveness.

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