HISTOLOGY OF THE PANCREAS IN DIABETES MELLITUS: "DEGENERATION" OF THE ISLETS OF LANGERHANS

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Abstract: This article provides a scientific analysis of histological changes in the pancreas in diabetes mellitus, with particular emphasis on the degeneration of the Islets of Langerhans. Diabetes mellitus, especially type 1, is characterized by autoimmune destruction of β-cells, islet atrophy, inflammatory infiltration, and fibrotic changes. In type 2 diabetes, amyloid deposition and progressive functional impairment are observed. These alterations lead to reduced insulin secretion and severe metabolic disturbances in the organism. The findings confirm that structural and functional damage of the Islets of Langerhans represents a central mechanism in the pathogenesis of diabetes mellitus.

Keywords: diabetes mellitus, pancreas, Islets of Langerhans, β-cells, insulitis, fibrosis, amyloid.

Introduction. Diabetes mellitus is one of the most complex and prevalent chronic endocrine disorders, characterized by impaired insulin secretion, reduced insulin action, or a combination of both. It represents a major global public health challenge, with a continuously increasing incidence and significant contribution to morbidity and mortality due to cardiovascular, renal, and neurological complications.

The endocrine portion of the pancreas-the Islets of Langerhans-serves as the central morphofunctional unit in the pathogenesis of diabetes mellitus. Within these islets, β-cells are responsible for insulin production, thereby regulating glucose metabolism and maintaining energy homeostasis. In addition, α-, δ-, and PP-cells contribute to the neuroendocrine regulation of glucose balance through the secretion of glucagon, somatostatin, and pancreatic polypeptide.

Type 1 diabetes mellitus is primarily an autoimmune disease in which T-lymphocytes mediate the destruction of β-cells, leading to absolute insulin deficiency. Histologically, this process is characterized by insulitis, defined as lymphocytic infiltration of the islets. In type 2 diabetes, insulin resistance initially leads to

compensatory β-cell hyperactivity, followed by progressive functional exhaustion and apoptosis.

Under chronic hyperglycemic conditions, progressive degenerative changes develop within the Islets of Langerhans, including reduction of β-cell mass, islet atrophy, increased connective tissue proliferation (fibrosis), and in some cases deposition of islet amyloid polypeptide (IAPP). These alterations further impair insulin secretion and exacerbate metabolic dysregulation.

Therefore, the study of histological alterations in the pancreas, particularly the degenerative processes of the Islets of Langerhans, is of significant scientific importance for understanding the pathogenesis of diabetes mellitus and for developing novel therapeutic strategies.

Main Part: The pancreas is a mixed gland with a complex morphofunctional structure consisting of both exocrine and endocrine components. The exocrine portion forms the major part of the gland and is composed of acinar cells responsible for the secretion of digestive enzymes such as amylase, lipase, and trypsinogen. The endocrine portion is represented by the Islets of Langerhans, which are diffusely distributed within the pancreatic parenchyma and serve as the principal neuroendocrine units responsible for maintaining glucose homeostasis.

Within the Islets of Langerhans, several distinct cell populations are identified, among which β-cells play a central role. β-cells synthesize and secrete insulin, the key hormone responsible for lowering blood glucose levels. α-cells produce glucagon, which increases blood glucose concentration, while δ-cells secrete somatostatin, which inhibits endocrine secretion. PP-cells produce pancreatic polypeptide that modulates gastrointestinal function. These cells interact through a highly coordinated neuroendocrine network to maintain glucose balance.

In diabetes mellitus, particularly type 1 diabetes, significant structural and functional alterations occur in the endocrine pancreas. Autoimmune mechanisms involving CD4+ and CD8+ T-lymphocytes target β-cells, leading to their progressive destruction. Histologically, this process is characterized by insulitis, defined as lymphocytic infiltration of the Islets of Langerhans. The loss of β-cells results in a marked reduction in insulin secretion and ultimately absolute insulin deficiency.

As the disease progresses, islet atrophy develops, characterized by a reduction in islet size, decreased cellularity, and diminished functional activity. Chronic hyperglycemia further promotes collagen deposition within the islet stroma, leading to fibrosis. This process compresses endocrine cells, disrupts trophic support, and further impairs secretory function.

In type 2 diabetes mellitus, the primary pathological mechanism is insulin resistance. Initially, β-cells undergo compensatory hypersecretion; however, over time, functional exhaustion and apoptosis occur. At this stage, degenerative changes are accompanied by deposition of islet amyloid polypeptide (IAPP) within the islets. These amyloid aggregates damage cell membranes, enhance oxidative stress, and accelerate β-cell death.

Additionally, inflammatory mediators such as IL-1β, TNF-α, and IFN-γ exacerbate β-cell destruction and induce oxidative stress and mitochondrial dysfunction within the local microenvironment. As a result, insulin secretion is further reduced, leading to worsening metabolic disturbances.

Overall, structural and functional damage of the Islets of Langerhans represents a central mechanism in the pathogenesis of diabetes mellitus. These histological changes form the morphological basis of insulin deficiency, hyperglycemia, lipid metabolism disorders, ketoacidosis in type 1 diabetes, and metabolic syndrome in type 2 diabetes.

Conclusion. Histological alterations of the pancreas in diabetes mellitus, particularly the degeneration of the Islets of Langerhans, represent one of the central morphofunctional mechanisms in the pathogenesis of the disease. Research indicates that in type 1 diabetes mellitus, autoimmune processes lead to progressive destruction of β-cells and, in the context of insulitis, result in absolute insulin deficiency. In type 2 diabetes mellitus, insulin resistance is accompanied by β-cell functional exhaustion, amyloid deposition, and chronic degenerative changes.

Atrophy, fibrosis, and cellular loss within the Islets of Langerhans lead to a marked reduction in insulin secretion, resulting in severe metabolic disturbances in the organism. Therefore, the study of the endocrine pancreas morphology is of significant scientific importance for understanding the pathogenesis of diabetes mellitus, improving early diagnostic approaches, and developing novel therapeutic strategies.

Overall, structural and functional impairment of the Islets of Langerhans constitutes a central event in the development of diabetes mellitus, making its investigation a highly relevant field in modern endocrinology and pathological anatomy.

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