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HISTOLOGICAL CHARACTERISTICS OF PANCREATIC ISLETS IN DIABETES MELLITUS

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Introduction

The pancreas is an important organ of the digestive and endocrine systems. Its endocrine component is represented by the islets of Langerhans, which contain several types of hormone-producing cells, including insulin-secreting beta cells, glucagon-secreting alpha cells, somatostatin-producing delta cells, and pancreatic polypeptide-producing cells. Beta cells play a central role in maintaining glucose homeostasis through insulin secretion. Diabetes mellitus is associated with structural and functional alterations of pancreatic islets, particularly changes affecting beta-cell mass and function. Histological investigation of the pancreatic endocrine apparatus provides important information about the morphological mechanisms underlying the development and progression of diabetes mellitus.

Relevance of the Problem

Diabetes mellitus is one of the most common chronic metabolic diseases and represents a major global health problem. Persistent hyperglycemia can result in cardiovascular, renal, neurological, and ophthalmological complications. Although the clinical manifestations of diabetes are well studied, investigation of the morphological changes occurring in pancreatic islets remains important for understanding the pathogenesis of the disease.

In type 1 diabetes mellitus, autoimmune destruction of pancreatic beta cells leads to severe insulin deficiency. In type 2 diabetes mellitus, insulin resistance is accompanied by progressive beta-cell dysfunction and changes in islet structure. Therefore, studying the histological characteristics of pancreatic islets may contribute to a deeper understanding of the mechanisms of diabetes and the development of approaches aimed at preservation or restoration of beta-cell function.

Methods

This thesis was prepared using a narrative review of scientific literature concerning the histological and morphological changes of pancreatic islets in diabetes mellitus. Scientific studies devoted to the structure of the islets of Langerhans, beta-cell morphology, insulin-producing cells, autoimmune insulitis, amyloid deposition, beta-cell apoptosis, and pancreatic tissue remodeling were analyzed.

Particular attention was paid to differences between type 1 and type 2 diabetes mellitus and to the principal microscopic changes observed in the endocrine portion of the pancreas.

Results

The analysis demonstrated that type 1 and type 2 diabetes mellitus are associated with different but partially overlapping histological alterations of the pancreatic islets.

In type 1 diabetes mellitus, one of the major histological features is a marked reduction in the number and mass of insulin-producing beta cells. During active autoimmune destruction, inflammatory infiltration of pancreatic islets, known as insulitis, may be observed. The inflammatory infiltrate predominantly consists of lymphocytes and is associated with progressive destruction of beta cells.

As the disease progresses, many pancreatic islets become smaller and contain markedly fewer insulin-positive cells. Alpha and delta cells may be relatively preserved compared with beta cells. Consequently, the normal cellular organization of the pancreatic islets becomes altered.

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In type 2 diabetes mellitus, complete destruction of beta cells is not usually observed. Instead, progressive beta-cell dysfunction and a reduction in functional beta-cell mass develop. Histological studies frequently demonstrate abnormalities in islet size and shape, beta-cell degeneration, cellular apoptosis, and alterations in the normal distribution of endocrine cells.

One of the characteristic morphological findings in type 2 diabetes mellitus is deposition of amyloid within pancreatic islets. Islet amyloid is largely associated with islet amyloid polypeptide, which is normally secreted together with insulin by beta cells. Excessive accumulation of amyloid may disturb normal islet architecture, impair cell-to-cell interactions, and contribute to progressive beta-cell dysfunction and death.

Chronic hyperglycemia and increased metabolic demand may also promote oxidative stress, mitochondrial dysfunction, and endoplasmic reticulum stress within beta cells. At the histological level, these pathological processes may be associated with cellular degeneration, reduced beta-cell density, and progressive remodeling of the endocrine pancreatic tissue.

Changes in the pancreatic microvasculature may additionally influence islet function. Because pancreatic islets are highly vascularized, abnormalities in capillary structure and blood supply may negatively affect oxygen delivery, hormone secretion, and the survival of endocrine cells.

Overall, the morphological findings indicate that diabetes mellitus involves not only disturbances in glucose metabolism but also significant structural alterations of the endocrine pancreas.

Conclusion

Diabetes mellitus is associated with characteristic histological changes in the pancreatic islets of Langerhans. Type 1 diabetes mellitus is predominantly characterized by autoimmune inflammation, insulitis, and progressive destruction of insulin-producing beta cells. Type 2 diabetes mellitus is associated with beta-cell dysfunction, reduction of functional beta-cell mass, cellular degeneration, apoptosis, and amyloid deposition within pancreatic islets.

Histological examination of pancreatic tissue contributes to understanding the cellular mechanisms involved in the development and progression of diabetes mellitus. Further investigation of beta-cell regeneration, preservation of pancreatic islet architecture, and prevention of endocrine cell damage may provide important perspectives for the development of new therapeutic strategies for diabetes.