Morphological changes in the pancreas in type 1 diabetes mellitus

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Abstract: Type 1 diabetes mellitus is an autoimmune disease characterized by selective destruction of pancreatic β-cells, resulting in absolute insulin deficiency. The principal morphological feature of early disease is insulitis, defined by lymphocytic infiltration of the islets of Langerhans. Progressive loss of β-cells leads to a reduction in islet size and number and the formation of pseudoatrophic islets with absent insulin expression. Immunohistochemical studies demonstrate preservation of non-β endocrine cells. The exocrine pancreas may also exhibit acinar atrophy and interstitial fibrosis. These morphological changes reflect the autoimmune pathogenesis of type 1 diabetes mellitus.

Keywords: Type 1 diabetes mellitus; pancreas; pancreatic morphology; histology; histopathology; islets of Langerhans; β-cell destruction; insulitis; autoimmune inflammation; immunohistochemistry; pancreatic atrophy.

Introduction: Type 1 diabetes mellitus (T1DM) is a chronic autoimmune disease characterized by immune-mediated destruction of insulin-producing β-cells in the pancreatic islets of Langerhans, resulting in absolute insulin deficiency and persistent hyperglycemia. Understanding the structural basis of this process is essential for elucidating disease pathogenesis and improving diagnostic and therapeutic strategies.

Morphological and histological studies have demonstrated that the hallmark feature of early-stage T1DM is insulitis, defined by lymphocytic infiltration of pancreatic islets, followed by progressive β-cell loss and formation of pseudoatrophic islets with absent insulin expression. In addition to endocrine alterations, the exocrine pancreas may also exhibit acinar atrophy and interstitial fibrosis, indicating that pancreatic involvement in T1DM extends beyond the islets and reflects a broader organ-level pathology.

Materials and Methods: This study was conducted as a descriptive histological analysis of pancreatic tissue from individuals with type 1 diabetes mellitus and non-diabetic controls. Pancreatic samples were obtained from autopsy material in accordance with ethical standards and institutional guidelines.

Tissue specimens were fixed in 10% neutral buffered formalin, processed using standard paraffin-embedding techniques, and sectioned at 4–5 μm thickness. Routine histological evaluation was performed using hematoxylin and eosin staining. Masson's trichrome staining was applied to assess fibrotic changes, and periodic acid–Schiff staining was used to evaluate basement membrane alterations.

Histological sections were examined by light microscopy. The morphology of the islets of Langerhans, including their size, number, and cellular integrity, as well as the presence of insulitis, β-cell depletion, acinar atrophy, fibrosis, and inflammatory infiltration, were assessed semi-quantitatively. Descriptive statistical analysis was applied where appropriate, with results expressed as mean ± standard deviation.

Results

Histological examination of pancreatic tissue from individuals with type 1 diabetes mellitus (T1DM) revealed profound alterations in both the endocrine and exocrine compartments compared to non-diabetic controls. The most prominent changes

were observed in the islets of Langerhans. There was a marked reduction in both the number and size of islets, and many islets displayed a pseudoatrophic appearance, characterized by the near-complete loss of β-cells. Non-β endocrine cells, including α- and δ-cells, were relatively preserved, leading to an altered cellular composition of the islets. The degree of β-cell depletion correlated with disease duration, with samples from individuals with recent-onset T1DM showing more prominent lymphocytic infiltration (insulitis). Lymphocytic infiltration was mainly perivascular and peri-islet, consisting predominantly of small mononuclear cells, consistent with autoimmune-mediated destruction. In long-standing cases, insulitis was less pronounced, reflecting the progressive loss of β-cells over time.

In the exocrine pancreas, morphological changes were also evident. Acinar atrophy was commonly observed, with reduction in cell size and occasional loss of acinar units. Interstitial fibrosis varied from mild to moderate, with connective tissue proliferation surrounding ducts and islets. Mild inflammatory infiltration was present around small ducts and acinar tissue. Occasional vascular alterations were detected, including thickening of capillary basement membranes and focal hyaline changes in small vessels. No evidence of necrosis or fatty infiltration was observed in most samples. These combined endocrine and exocrine alterations indicate that T1DM affects the pancreas at the organ level, not only through selective β-cell destruction but also via secondary changes in surrounding tissue.

Overall, the histological findings demonstrate a consistent pattern of autoimmune-mediated β-cell loss, insulitis in early disease, and associated structural changes in the exocrine pancreas. These observations underscore the importance of considering both endocrine and exocrine pathology when studying the morphological consequences of T1DM.

Conclusion: Type 1 diabetes mellitus is associated with profound morphological changes in the pancreas, primarily affecting the endocrine compartment.

Histological analysis demonstrates marked reduction in the number and size of islets of Langerhans, pseudoatrophic appearance of islets due to β-cell loss, and lymphocytic infiltration (insulitis) in early disease stages. Non-β endocrine cells are relatively preserved, whereas the exocrine pancreas exhibits acinar atrophy, interstitial fibrosis, and mild inflammatory infiltration. These findings indicate that pancreatic pathology in T1DM extends beyond the islets and reflects the autoimmune-mediated destruction of β-cells, contributing to both functional impairment and structural remodeling of the organ. Understanding these morphological alterations provides a foundation for further research into disease mechanisms and potential therapeutic strategies.

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