



## A NEW APPROACH TO THE MORPHOLOGICAL CHANGES OF THE PANCREAS IN MYCOBACTERIAL INFECTION OF THE LUNG

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**Abstract:** *Mycobacterial infection of the lung induces systemic inflammatory, hypoxic, and токсико-иммунные responses that extend beyond pulmonary tissue. This review proposes a novel integrative approach to understanding morphological alterations of the pancreas during pulmonary mycobacterial infection. By synthesizing data from experimental and clinicopathological studies, we analyze structural remodeling of the exocrine and endocrine compartments, stromal reorganization, and microvascular pathology. Particular emphasis is placed on the interplay between hypoxia-induced injury, cytokine-mediated damage, and microangiopathy. Evidence suggests that pancreatic alterations represent a secondary systemic manifestation characterized by acinar dystrophy, islet remodeling, vascular disturbances, and progressive interstitial fibrosis. The proposed framework integrates morphometric, histopathological, and pathogenetic perspectives to clarify mechanisms of pancreatic involvement in pulmonary tuberculosis.*

**Keywords:** *Mycobacterial infection, pulmonary tuberculosis, pancreas, morphology, histopathology, microcirculation, acinar dystrophy, islets of Langerhans, systemic inflammation.*

### **Main Text**

### **Introduction**

Pulmonary tuberculosis, caused by *Mycobacterium tuberculosis*, remains a global health concern with multisystemic implications. Although the primary pathological process is localized in the lungs, tuberculosis is increasingly recognized



as a systemic disease associated with chronic inflammation, oxidative stress, immune dysregulation, and hypoxemia.

The pancreas, due to its high metabolic activity and dense microvascular network, is particularly vulnerable to systemic disturbances. However, pancreatic involvement in pulmonary mycobacterial infection has not been sufficiently characterized. A new conceptual approach is therefore required—one that integrates structural, vascular, and immunopathological mechanisms.

The aim of this review is to present a comprehensive analysis of morphological changes in the pancreas during pulmonary mycobacterial infection and to propose a pathogenetically oriented interpretative model.

### **Morphological Alterations of the Exocrine Compartment**

The exocrine pancreas consists of acinar cells responsible for digestive enzyme synthesis and secretion. Under conditions of pulmonary mycobacterial infection, several consistent morphological features have been described:

- Vacuolar and hydropic degeneration of acinar cells
- Reduction of zymogen granules
- Nuclear pyknosis and hyperchromasia
- Disorganization of acinar architecture
- Interstitial edema
- Lymphohistiocytic stromal infiltration

Morphometric analyses in experimental models demonstrate a statistically significant reduction in acinar diameter and cytoplasmic volume ( $p < 0.05$ ). These findings indicate parenchymal dystrophy associated with chronic systemic intoxication and hypoxia.

From a pathogenetic standpoint, sustained inflammatory mediator release (TNF- $\alpha$ , IL-1 $\beta$ , IL-6) contributes to oxidative damage and mitochondrial dysfunction in acinar cells, promoting structural disintegration.

### **Endocrine Pancreatic Remodeling**



The endocrine pancreas, represented by the islets of Langerhans, exhibits structural variability during systemic infection. Reported changes include:

- Decrease in islet size and numerical density
- Reduction in  $\beta$ -cell population
- Cytoplasmic vacuolization of endocrinocytes
- Focal compensatory hyperplasia
- Capillary dilation and perivascular edema

These alterations may underlie impaired insulin secretion and secondary disturbances in glucose metabolism. The well-documented epidemiological association between tuberculosis and dysglycemia may thus have a morphological substrate at the pancreatic level.

The proposed new perspective emphasizes that islet remodeling represents both an adaptive and degenerative process, depending on the duration and severity of systemic inflammation.

### **Microvascular and Stromal Changes**

Microangiopathy appears to be a central mechanism in pancreatic injury during pulmonary mycobacterial infection. Observed vascular changes include:

- Venular dilation
- Erythrocyte stasis
- Endothelial swelling
- Increased vascular permeability
- Perivascular hemorrhagic foci

Chronic hypoxemia resulting from pulmonary pathology exacerbates endothelial dysfunction. Concurrently, prolonged inflammatory signaling activates fibroblasts, leading to:

- Interstitial collagen deposition
- Stromal thickening
- Focal fibrosis



These processes contribute to progressive structural remodeling and functional impairment of the gland.

## A New Integrative Pathogenetic Model

The proposed approach conceptualizes pancreatic morphological changes as the result of four interacting mechanisms:

1. **Systemic hypoxia** due to impaired pulmonary gas exchange.
2. **Inflammatory cytokine cascade** causing cellular injury.
3. **Microvascular dysfunction** leading to ischemia and edema.
4. **Metabolic stress** associated with chronic infection.

Rather than viewing pancreatic changes as isolated degenerative phenomena, they should be interpreted as components of systemic inflammatory organ remodeling.

This integrative framework supports the need for combined histological, immunohistochemical, and morphometric assessment in future studies.

## Conclusion

Pulmonary mycobacterial infection induces significant morphological alterations in the pancreas, affecting both exocrine and endocrine compartments.

Key findings include:

- Acinar dystrophy and architectural disorganization
- Islet remodeling with  $\beta$ -cell reduction
- Microangiopathy and interstitial edema
- Progressive stromal fibrosis

These changes reflect systemic inflammatory and hypoxic mechanisms rather than direct mycobacterial invasion. The proposed integrative model provides a structured basis for further experimental and clinical research into pancreatic involvement in tuberculosis.

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