ANALYSIS OF MORPHO-FUNCTIONAL CHANGES IN THE HEART IN EXPERIMENTAL MYCOBACTERIAL LUNG DAMAGE

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Abstract: *This study is devoted to a comparative analysis of morphological changes in cardiac tissue under conditions of experimental mycobacterial lung injury caused by Mycobacterium tuberculosis. The investigation was conducted using a laboratory animal model, in which histological and morphometric methods were applied to assess structural and morphofunctional remodeling of the myocardium during chronic pulmonary inflammation. It was established that mycobacterial lung damage is accompanied by the development of chronic hypoxia, systemic intoxication, and pulmonary hypertension, leading to dystrophic, microcirculatory, compensatory-hypertrophic, and fibrotic changes in the myocardium. The most pronounced alterations were observed in the right ventricle and were characterized by myocardial hypertrophy and the formation of interstitial fibrosis. The obtained data expand current understanding of the pathogenetic mechanisms underlying secondary cardiac involvement in pulmonary tuberculosis.*

Keywords: *mycobacteria, pulmonary tuberculosis, cardiac morphology, myocardium, hypoxia, pulmonary hypertension, right ventricular hypertrophy, interstitial fibrosis, experimental study.*

Main Body

Introduction

Pulmonary tuberculosis is a chronic infectious disease characterized by persistent inflammatory processes, immune dysregulation, and systemic intoxication. Mycobacterial involvement of the lung parenchyma adversely affects not only the respiratory system but also the cardiovascular system.

Granulomatous inflammation of the lungs, reduction of the alveolar respiratory surface, and impaired gas exchange contribute to the development of chronic hypoxia and increased vascular resistance in the pulmonary circulation. These changes result in pulmonary hypertension and a subsequent increase in hemodynamic load on the right chambers of the heart. Under conditions of prolonged overload, compensatory mechanisms are activated in the myocardium, which are later replaced by destructive structural alterations.

In this context, the study of cardiac morphological remodeling in experimental pulmonary tuberculosis is of significant relevance to modern morphology and pathophysiology.

Materials and Methods

The study was performed on 60 laboratory rats, which were divided into two groups:

- **Control group** – intact healthy animals;
- **Experimental group** – animals with intratracheal administration of *Mycobacterium tuberculosis*.

Observations were carried out at 30 and 60 days after infection. At the specified time points, animals were euthanized, and cardiac tissue samples were collected for morphological examination.

Histological sections were stained using:

- hematoxylin and eosin for general morphological assessment;
- Van Gieson staining for visualization of connective tissue and fibrosis.

Morphometric analysis included the evaluation of:

- cardiomyocyte diameter;
- myocardial thickness of the right and left ventricles;
- proportion of interstitial tissue;
- thickness of arteriolar walls.

Statistical analysis was performed using Student's *t*-test. Differences were considered statistically significant at $p < 0.05$.

Results

Control Group

In control animals, the heart exhibited normal histoarchitectonics. Myocardial fibers were regularly arranged, cardiomyocytes had clear contours and centrally located nuclei. Interstitial tissue was minimal, and vascular walls showed no pathological changes.

Experimental Group (30 Days)

At 30 days after mycobacterial infection, the following morphological alterations were observed in the myocardium:

- granular and vacuolar degeneration of cardiomyocytes;
- clarification of sarcoplasm;
- interstitial edema;
- venous congestion of capillaries and venules.

Morphometric analysis demonstrated a statistically significant increase in cardiomyocyte diameter compared with the control group ($p < 0.05$), indicating early compensatory responses to hypoxia.

Experimental Group (60 Days)

At 60 days, myocardial changes became more pronounced:

- thickening of the right ventricular wall (compensatory hypertrophy);
- cardiomyocyte hypertrophy with nuclear hyperchromasia;
- formation of interstitial fibrotic foci;
- disorganization of myofibrils;
- sclerotic changes in arteriolar walls.

The thickness of the right ventricular myocardium was significantly greater than in the control group ($p < 0.01$), while the proportion of interstitial connective tissue increased by 1.7–1.9 times.

Discussion

The obtained results indicate that mycobacterial lung injury induces systemic morphological remodeling of cardiac tissue. In the early stages, dystrophic and

microcirculatory disturbances predominate, primarily due to hypoxia and systemic intoxication. In later stages, compensatory-hypertrophic and fibrotic processes become dominant, associated with the development of pulmonary hypertension.

Right ventricular hypertrophy represents a morphological manifestation of chronic pressure overload, whereas interstitial fibrosis reduces myocardial elasticity and creates a structural basis for the development of heart failure.

Thus, the observed cardiac morphological changes reflect the systemic nature of pulmonary tuberculosis and its long-term impact on the cardiovascular system.

Conclusion

1. Experimental mycobacterial lung injury leads to early development of dystrophic and microcirculatory changes in the myocardium.
2. In later stages, right ventricular hypertrophy, interstitial fibrosis, and vascular sclerosis are formed.
3. Morphometric parameters confirm the statistical significance of the observed myocardial remodeling ($p < 0.05$).
4. The identified morphological alterations are pathogenetically associated with chronic hypoxia and pulmonary hypertension.

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