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Stress-Induced Secondary Immunodeficiency: Histological Changes in the Thymus and Lymph Nodes (Experimental Interpretation)

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Abstract: The immune system is a complex morphofunctional structure responsible for defending the body against internal and external antigens. With the rising prevalence of stress factors, secondary immunodeficiency conditions have become increasingly common. Experimental and clinical data demonstrate that stress-induced immunosuppression leads to distinct morphological alterations in lymphoid organs, particularly the thymus and lymph nodes. This article analyzes histological changes in these organs under stress-induced secondary immunodeficiency, based on theoretical sources and experimental models.

Key Words: stress, secondary immunodeficiency, thymus, lymph nodes, apoptosis, histology, glucocorticoids.

Introduction

The immune system regulates protective reactions against various endogenous and exogenous antigens. In recent years, the incidence of secondary immunodeficiency has risen due to increasing exposure to stressors. Psychological pressure, chronic inflammatory diseases, ecological factors, malnutrition, infections, and autoimmune processes negatively affect immune function. Stress-induced immunodepression is characterized by marked morphological alterations in the thymus and lymph nodes.

Neuroendocrine mechanisms, hormonal imbalance, apoptosis, and stromal destruction play a leading role in the pathogenesis of secondary immunodeficiency. Studying the morphological effects of stress provides valuable insight for clinical immunology, pathomorphology, and experimental medicine.

Aim

To analyze histological changes in the thymus and lymph nodes under stress-induced secondary immunodeficiency based on theoretical data and experimental observations.

Relevance

- Stress and immunodeficiency are interrelated pathological processes.
- The thymus, as the central organ of T-lymphocyte maturation, is highly stress-sensitive.
- Lymph nodes are the main peripheral targets of the immune response.
- Experimental findings hold clinical value.
- Chronic stress increases susceptibility to infections and autoimmune diseases.

Literature Review

Scientific publications describe the cytotoxic and apoptotic effects of glucocorticoids, adrenaline, and catecholamines on lymphoid tissues. Prolonged stress causes lymphocyte depletion in the thymic cortex, degeneration of Hassall's corpuscles, and fibro-sclerotic alterations. In lymph nodes, reduced follicular centers, stromal collapse, and lymphocyte depletion of sinuses have been reported.

Overview of Experimental Models

Stress-induced immunodeficiency in rodents is commonly studied through:

- Immobilization stress
- Cold exposure (4–6 °C)
- Food restriction or sleep deprivation
- Alteration of light and noise regimes
- Administration of high-dose corticosteroids

The duration typically ranges from 7 to 30 days, followed by comparison with control groups.

Methods of assessment:

- Hematoxylin–eosin staining
- Immunohistochemistry (CD3, CD4, CD8, caspase-3)
- Histochemistry (PAS-reaction, Van Gieson, Sudan III)
- Light, electron, and phase-contrast microscopy
- Histological Changes in the Thymus
- The following alterations have been identified under stress exposure:
- Lymphocytic necrosis and apoptosis in the cortical zone
- Reduced lymphocyte count and stromal destruction in the medulla
- Vacuolization, shrinkage, and wrinkling of Hassall's corpuscles
- Reduced thymocyte proliferation
- Microcirculatory disturbance: congestion, stasis, perivascular edema
- Fatty degeneration and atrophy
- Glucocorticoid-induced thymocyte apoptosis is considered the leading pathogenic mechanism.
- Histological Changes in Lymph Nodes

Stress-induced immunodepression causes:

- Shrinkage or disappearance of cortical follicles
- Decreased proliferative activity in germinal centers
- Signs of atrophy in the T-cell paracortex
- Slowed differentiation of plasma cells
- Early sclerotic changes in the reticular stroma
- Reduced macrophage content in sinuses
- Vascular stasis in veins and capillaries
- Replacement of follicles by adipose cells in some cases
- Relationship Between Stress and Immunodeficiency

Stress weakens the immune system through neuroendocrine mechanisms:

- Increased ACTH and cortisol secretion
- Catecholamine-induced lymphoid tissue regression
- Disruption of T-lymphocyte proliferation

- Enhanced apoptosis
- Depletion of lymphoid reserves

Consequently, the organism becomes more susceptible to infections, inflammatory disorders, and autoimmune diseases.

Discussion

Experimental findings indicate that histological changes in the thymus and lymph nodes depend on the intensity and duration of stress. Morphological analysis provides a basis for early diagnosis and immunoprophylaxis.

Conclusion

- Stress-induced immunodeficiency leads to structural and functional disturbances in lymphoid organs.
- The thymus exhibits atrophy, apoptosis, stromal destruction, and fatty degeneration.
- Lymph nodes show follicular regression, sclerosis, and reduction of immune cells.
- Experimental models are effective for understanding clinical immunopathology.
- Morphological research is critical for the prevention of stress-related immunodeficiency.

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