PATHOMORPHOLOGICAL CHANGES IN THE THYMUS AND LYMPH NODES OF NEWBORNS WHO DIED FROM ASSISTED INTRAUTERINE VIRAL PNEUMONIA

Ochilov Sardor

Samarkand State Medical University, Department of Pathological Anatomy and Sectional Biopsy, 1st-year Master's Resident

Scientific supervisor: **Ismoilov Jasur Mardonovich**

Abstract: This article is devoted to the study of pathomorphological changes in the thymus and lymph nodes of newborns who died from intrauterine viral pneumonia. The results of the study revealed cortical atrophy, lymphocyte depletion, and disruption of the cortical-medullary architecture in the thymus, as well as follicular atrophy, sinus histiocytosis, and vascular changes in the lymph nodes. These pathological alterations reduce the efficiency of the neonatal immune system, increase susceptibility to infections, and worsen clinical outcomes. The findings provide important scientific insights into the pathogenesis of intrauterine viral pneumonia and neonatal immunopathology, contributing to the development of preventive and therapeutic strategies.

Keywords: Intrauterine viral pneumonia, Newborn, Thymus, Lymph nodes, Pathomorphological changes, Neonatal immunity, Lymphocyte depletion, Immunopathology.

Introduction

Assisted viral intrauterine pneumonia is a severe neonatal disease that develops as a result of viral infection transmitted during pregnancy. This condition is associated with a high mortality rate in newborns and leads to significant pathological changes in the development of the immune system. Neonatal pneumonia of viral origin remains a global health problem, as it substantially affects the health and survival of newborns. In particular, in newborns whose immune system is not yet fully developed, intrauterine viral infections can cause structural and functional changes in primary and secondary lymphoid organs, including the thymus and lymph nodes. The thymus and lymph nodes are key

organs of the immune system, responsible for the differentiation and maturation of lymphocytes and the development of effective immune responses. Viral infections during intrauterine life can induce profound pathological morphologic changes in these organs, including atrophy of the thymic cortex, depletion of lymphocytes, disruption of normal lymphoid follicle architecture, and other structural alterations in the lymph nodes. Understanding these changes is crucial because they directly influence the neonate's ability to respond to infectious agents and develop long-term immune competence.

Relevance

Intrauterine viral pneumonia is a significant cause of neonatal morbidity and mortality. Newborns with an underdeveloped immune system are particularly vulnerable to viral infections acquired during pregnancy. These infections can cause structural and functional changes in the thymus and lymph nodes, impairing lymphocyte development and reducing immune competence. Understanding these pathological changes is important for clarifying the mechanisms of neonatal immune dysfunction and improving preventive and therapeutic strategies.

Objective

The main objective of this study is to investigate the pathological morphological changes in the thymus and lymph nodes of newborns who died from intrauterine viral pneumonia. Specifically, the study aims to describe structural alterations in these organs and assess their impact on neonatal immune system development.

Main part

Intrauterine viral pneumonia represents a significant neonatal health issue worldwide. Epidemiological studies indicate that viral infections acquired during pregnancy are among the leading causes of neonatal morbidity and mortality. The prevalence of these infections varies according to geographic regions, maternal health, and access to prenatal care. Maternal viral infections, such as cytomegalovirus, respiratory syncytial virus, and influenza, can cross the placental barrier and infect the fetus, leading to intrauterine pneumonia. Newborns born

prematurely or with low birth weight are particularly susceptible due to their underdeveloped immune systems. Epidemiological data also suggest that socioeconomic factors, maternal immunization status, and the presence of coexisting maternal diseases significantly influence the incidence and severity of intrauterine viral pneumonia. Monitoring and understanding these patterns are crucial for developing effective preventive strategies and for allocating healthcare resources efficiently. Additionally, global data emphasize the importance of early maternal screening and vaccination programs to reduce viral transmission rates and neonatal complications.

Viral intrauterine pneumonia arises primarily from maternal viral infections that are transmitted to the fetus through the placenta or during labor. The most common etiological agents include cytomegalovirus, adenovirus, herpes simplex virus, parainfluenza viruses, and respiratory syncytial virus. Pathogenesis involves viral replication in the fetal respiratory tract, triggering inflammatory responses that damage pulmonary tissue. Viral particles can induce apoptosis of epithelial cells and disrupt the normal lung development. Additionally, viral infections provoke systemic immune responses, leading to infiltration of immune cells into the lungs and secondary organs, including the thymus and lymph nodes. These changes result in impaired lymphocyte maturation, altered cytokine profiles, and reduced adaptive immune function. Understanding the mechanisms by which viruses affect fetal organs is critical for developing targeted antiviral therapies and for predicting the clinical course in affected newborns.

The thymus is a primary lymphoid organ responsible for T-lymphocyte development and immune system maturation. In neonates, the thymus is relatively large and highly active, with distinct cortical and medullary regions that support lymphocyte differentiation and selection. Viral infections during intrauterine life can significantly alter thymic architecture, leading to cortical atrophy, medullary disruption, and lymphocyte depletion. These pathological changes compromise the thymus’s ability to produce competent T-cells, which are essential for adaptive immunity. Morphological examination often reveals reduced thymic size, altered

Hassall's corpuscles, and irregular cortical-medullary boundaries. The functional consequences include impaired immune responses, increased susceptibility to infections, and potential long-term immune dysregulation. Studying these changes provides critical insights into the neonatal immunopathology associated with viral intrauterine pneumonia.

Secondary lymphoid organs, particularly lymph nodes, play a vital role in initiating immune responses and maintaining lymphocyte homeostasis. In neonates affected by viral intrauterine pneumonia, lymph nodes often demonstrate structural disorganization. Histopathological analysis can reveal lymphoid follicle atrophy, depletion of B and T lymphocytes, sinus histiocytosis, and vascular congestion. These changes reduce the lymph nodes' capacity to mount effective immune responses and facilitate systemic viral dissemination. Additionally, chronic inflammation may lead to fibrosis or stromal disruption, further impairing lymphocyte trafficking. Understanding these morphological alterations helps to explain the compromised immune competence observed in affected newborns and can guide therapeutic interventions aimed at supporting lymphoid organ function.

Viral intrauterine infections induce significant immune system dysfunction in neonates. Altered thymic and lymph node structures disrupt lymphocyte maturation and differentiation, leading to reduced T-cell and B-cell populations. Cytokine imbalance, increased inflammatory mediators, and impaired antigen presentation further exacerbate immune dysregulation. Clinically, affected newborns demonstrate increased susceptibility to secondary infections, delayed immune responses, and poor vaccine responsiveness. Evaluating the extent of immune dysfunction in these neonates is essential for predicting clinical outcomes and developing targeted immunomodulatory therapies. Comprehensive studies of immune parameters, including lymphocyte subsets, serum immunoglobulins, and cytokine profiles, provide valuable information about the severity of immune impairment.

Neonates with intrauterine viral pneumonia present with a wide spectrum of clinical symptoms, including respiratory distress, cyanosis, apnea, fever, and

feeding difficulties. The severity of symptoms often correlates with gestational age, viral load, and the extent of immune system impairment. Laboratory findings may show leukopenia, lymphopenia, and elevated inflammatory markers. Radiological imaging often reveals bilateral pulmonary infiltrates and interstitial pneumonia patterns. Unfortunately, severe cases may result in neonatal death, particularly when the infection occurs in the late second or third trimester and affects multiple organ systems. Long-term outcomes for survivors may include chronic lung disease, impaired growth, and persistent immune deficits. Understanding clinical features alongside pathomorphological findings is essential for accurate diagnosis and management. Accurate diagnosis of viral intrauterine pneumonia relies on a combination of clinical evaluation, laboratory testing, and histopathological analysis. Polymerase chain reaction testing, viral cultures, and serological assays help identify the causative virus. Histopathological examination of the thymus and lymph nodes provides direct evidence of organ-specific pathological changes. Tissue samples are typically stained and examined under light microscopy to assess lymphocyte density, cortical-medullary architecture, follicular integrity, and the presence of viral inclusions. Immunohistochemical techniques may be used to detect specific viral antigens and lymphocyte markers, providing insight into the immune response and viral localization. Comprehensive diagnostic approaches allow for correlation between clinical presentation and underlying pathological changes, facilitating more targeted treatment strategies.

Understanding the pathomorphological changes in the thymus and lymph nodes has direct implications for the prevention and treatment of viral intrauterine pneumonia. Early maternal screening for viral infections, vaccination programs, and antiviral therapies during pregnancy are essential to reduce fetal infection risk. In affected neonates, supportive care, immunomodulatory treatments, and close monitoring of immune function are critical. Knowledge of specific structural and functional alterations in lymphoid organs may inform future therapies aimed at restoring immune competence. Additionally, research findings can contribute to the

development of evidence-based guidelines for neonatal care, ultimately improving survival rates and long-term health outcomes in this vulnerable population.

Discussion

The findings of this study demonstrate that intrauterine viral pneumonia induces severe pathomorphological changes in the thymus and lymph nodes, which are key organs of the immune system. Cortical atrophy and lymphocyte depletion in the thymus indicate impaired T-cell maturation, which may lead to weakened adaptive immunity and increased susceptibility to infections in affected neonates. These results are consistent with previous research emphasizing the vulnerability of the neonatal immune system to prenatal viral infections. Structural alterations in the lymph nodes, including follicular atrophy, sinus histiocytosis, and vascular changes, further reflect systemic immune impairment. The reduction in lymphocyte populations and stromal disruptions compromise antigen recognition and lymphocyte trafficking, making neonates less capable of mounting effective immune responses. The presence of viral antigens in lymphoid tissue suggests that direct viral replication contributes to organ damage, while secondary inflammation exacerbates tissue injury. Clinically, the observed pathomorphological changes correlate with severe respiratory symptoms and laboratory abnormalities in affected neonates. These findings underline the importance of early detection and prevention of maternal viral infections during pregnancy, as well as the need for targeted neonatal care and immune support. Furthermore, understanding these structural changes provides insight into the mechanisms of immune dysfunction, informing the development of novel therapeutic and preventive strategies to improve neonatal survival and long-term health outcomes. This study emphasizes that intrauterine viral pneumonia not only damages pulmonary tissue but also severely disrupts the development and function of thymus and lymph nodes. Such pathological changes compromise immune competence in neonates, underscoring the critical need for preventive measures, early diagnosis, and specialized care for affected newborns.

Results

In this study, the pathological examination of the thymus and lymph nodes in newborns who died from intrauterine viral pneumonia revealed significant structural and functional alterations. The thymus showed marked cortical atrophy, reduced density of lymphocytes, and irregularities in the cortical-medullary architecture. Hassall's corpuscles were often distorted or decreased in number, reflecting impaired thymic maturation. These structural changes suggest a profound disruption in T-cell development, which is critical for effective adaptive immunity in neonates. Lymph nodes exhibited follicular atrophy, depletion of B and T lymphocytes, sinus histiocytosis, and vascular congestion. Some samples demonstrated stromal disruption and mild fibrosis, indicating chronic inflammatory changes. Immunohistochemical analysis revealed decreased expression of key lymphocyte markers and occasional presence of viral antigens, confirming the direct viral impact on lymphoid tissue. Clinically, affected neonates presented with respiratory distress, cyanosis, and feeding difficulties, consistent with severe viral pneumonia. Laboratory findings showed leukopenia and lymphopenia, while radiological imaging indicated bilateral interstitial infiltrates in the lungs. These results collectively highlight that intrauterine viral infections profoundly affect both primary and secondary lymphoid organs, compromising the neonatal immune system and contributing to poor clinical outcomes.

Conclusion

The study demonstrates that intrauterine viral pneumonia causes profound pathological changes in both the thymus and lymph nodes of affected newborns. Thymic alterations, including cortical atrophy, lymphocyte depletion, and disrupted cortical-medullary architecture, indicate severe impairment of T-cell development and adaptive immunity. Similarly, lymph node changes, such as follicular atrophy, sinus histiocytosis, and vascular congestion, further compromise the neonate's immune competence. These findings confirm that viral infections acquired during intrauterine life not only damage pulmonary tissue but also significantly affect the primary and secondary lymphoid organs, thereby increasing the susceptibility of newborns to infections and contributing to poor clinical outcomes. Understanding

these structural and functional changes provides crucial insights into the immunopathology of neonatal viral pneumonia. The results of this research underscore the importance of preventive strategies, including maternal viral screening, vaccination, and antiviral interventions during pregnancy. For affected neonates, early diagnosis, intensive monitoring, and supportive immunomodulatory therapy are essential to improve survival rates and long-term health outcomes. Overall, this study highlights the critical relationship between prenatal viral infections and neonatal immune system development, offering a foundation for future research and clinical interventions aimed at reducing morbidity and mortality in this vulnerable population.

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