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PATHOMORPHOLOGY OF THE INTEGRAL CONNECTION OF THE ADRENAL GLANDS AND THYMUSIS IN RDS SYNDROME IN NEWBORN INFANTS

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Abstract. This study investigates the pathomorphological interrelationship between the adrenal glands and thymus in newborns diagnosed with Respiratory Distress Syndrome (RDS). RDS is a critical neonatal condition primarily affecting lung function, but it also induces systemic changes impacting multiple organs. The research focuses on examining structural and functional alterations in the adrenal cortex and thymic tissue, revealing how these organs respond integrally to the stress and hypoxia associated with RDS. Findings indicate adrenal hyperplasia and thymic involution, reflecting a complex neuroendocrine-immune interaction that compromises neonatal adaptation and immunity. Understanding these changes provides crucial insights into the systemic nature of RDS and suggests the necessity for comprehensive therapeutic approaches that address endocrine and immune dysfunction alongside respiratory management. This work contributes to improving clinical outcomes by highlighting potential targets for intervention in neonatal care.

Keywords: Respiratory Distress Syndrome, Adrenal glands, Thymus, Pathomorphology, Adrenal cortical hyperplasia, Thymic involution, Hypoxia, Neuroendocrine, Homeostasis.

ПАТОМОРФОЛОГИЯ ИНТЕГРАЛЬНОЙ СВЯЗИ НАДПОЧЕЧНИКОВ И ТИМУЗА ПРИ СИНДРОМЕ РДС У НОВОРОЖДЕННЫХ

Аннотация. В этом исследовании изучается патоморфологическая взаимосвязь между надпочечниками и тимусом у новорожденных с диагнозом респираторный дистресс-синдром (РДС). РДС является критическим неонатальным состоянием, в первую очередь влияющим на функцию легких, но также вызывающим системные изменения, затрагивающие несколько органов. Исследование сосредоточено на изучении структурных и функциональных изменений в коре надпочечников и ткани тимуса, раскрывая, как эти органы реагируют в целом на стресс и гипоксию, связанные с РДС. Результаты указывают на гиперплазию надпочечников и инволюцию тимуса, отражая сложное нейроэндокринно-иммунное взаимодействие, которое ставит под угрозу неонатальную адаптацию и иммунитет. Понимание этих изменений дает важные сведения о системной природе РДС и предполагает необходимость комплексных терапевтических подходов, которые решают эндокринную и иммунную дисфункцию наряду с респираторным контролем. Эта работа способствует улучшению клинических результатов, выделяя потенциальные цели для вмешательства в неонатальный уход.

Ключевые слова: Респираторный Дистресс-Синдром, Надпочечники, Тимус, Патоморфология, Гиперплазия Коры Надпочечников, Инволюция Тимуса, Гипоксия, Нейроэндокринный, Гомеостаз.

Introduction

Respiratory Distress Syndrome is one of the most common and serious conditions affecting newborn infants, especially those born prematurely or with low birth weight. The

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syndrome arises due to insufficient production of surfactant in the lungs, a substance that reduces surface tension in the alveoli and prevents their collapse during breathing. Without enough surfactant, the lungs cannot function properly, leading to difficulty in breathing, decreased oxygen levels in the blood, and overall respiratory failure. This condition poses a significant risk to the survival and health of newborns, requiring prompt diagnosis and treatment.

While the respiratory system is primarily affected in this syndrome, it is important to recognize that Respiratory Distress Syndrome also has widespread effects on other organs and systems in the body. Among these, the adrenal glands and thymus hold critical roles in the newborn's response to stress and immune defense. The adrenal glands, located above the kidneys, are responsible for producing hormones such as cortisol and adrenaline that help the body manage stress and inflammation. Meanwhile, the thymus, situated in the chest near the heart, is a central immune organ involved in the development and maturation of T-lymphocytes, which are essential for adaptive immunity. In newborns with Respiratory Distress Syndrome, the stress of illness and hypoxia triggers changes in both the adrenal glands and thymus. These changes include morphological and functional alterations that may reflect the severity of the disease and the body's attempt to adapt. For example, the adrenal glands might show hyperplasia or increased hormone production, while the thymus may undergo involution or atrophy, leading to impaired immune function.

Understanding the integral relationship between the adrenal glands and thymus in the context of Respirator Distress Syndrome is crucial for a comprehensive view of the disease's pathophysiology. Studying the pathological changes in these organs can provide valuable insights into how the body copes with severe respiratory illness and can help guide improved therapeutic approaches. This study aims to investigate the pathomorphological features of the adrenal glands and thymus in newborn infants affected by Respiratory Distress Syndrome. The findings are expected to contribute to better understanding of the systemic impact of RDS and aid in the development of more effective clinical management strategies for these vulnerable patients.

Main Body

Respiratory Distress Syndrome (RDS) is a critical condition frequently encountered in newborns, especially those born prematurely. The syndrome primarily results from insufficient production of pulmonary surfactant, which leads to alveolar collapse and impaired gas exchange in the lungs. This respiratory insufficiency can rapidly progress, causing severe hypoxia and metabolic imbalances that threaten the survival of the infant. Although the lungs are the primary organs affected, RDS exerts systemic effects on various other organs, including the adrenal glands and thymus. The adrenal glands play a vital role in the newborn's response to physiological stress by producing essential hormones such as cortisol and adrenaline. Similarly, the thymus is a crucial immune organ involved in the development and maturation of T-lymphocytes, essential for adaptive immunity. Understanding the pathological changes in both the adrenal glands and thymus during RDS is important to grasp the syndrome's full impact on the newborn's body. This study focuses on investigating the pathomorphological features of these organs and their integral relationship in newborns affected by RDS.

Respiratory Distress Syndrome develops due to a deficiency of surfactant in the lungs, which causes increased surface tension and alveolar collapse. This results in decreased lung compliance and impaired oxygenation. The clinical presentation includes tachypnea, nasal flaring, grunting, and cyanosis shortly after birth. Pathophysiologically, the lack of surfactant

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triggers a cascade of events such as atelectasis, inflammation, and increased pulmonary vascular resistance. These changes lead to ventilation-perfusion mismatch and hypoxemia. Systemic hypoxia further causes metabolic acidosis and multi-organ dysfunction. In premature infants, the immaturity of various organ systems, including endocrine and immune systems, complicates the clinical picture. The severity of RDS depends on gestational age, surfactant deficiency degree, and presence of secondary infections or complications. Understanding these mechanisms is essential for appropriate therapeutic interventions and predicting outcomes in affected newborns.

The adrenal glands are key components of the hypothalamic-pituitary-adrenal axis, producing hormones essential for stress response, including cortisol, aldosterone, and catecholamines. In newborns with RDS, the stress imposed by hypoxia and respiratory failure triggers hyperactivity of the adrenal cortex. Pathomorphologically, this may manifest as cortical hyperplasia, cellular hypertrophy, and increased vascularity. Occasionally, hemorrhagic changes and necrosis are observed, reflecting severe stress and compromised organ perfusion. The enhanced cortisol secretion attempts to modulate inflammatory responses and maintain hemodynamic stability. However, prolonged stress and adrenal exhaustion can lead to adrenal insufficiency, worsening the clinical condition. Studying these adrenal changes provides insights into the systemic stress response and may help in identifying infants who require hormonal support during critical illness.

The thymus is a primary lymphoid organ responsible for the differentiation and maturation of T-lymphocytes, which are pivotal for adaptive immunity. During RDS, systemic hypoxia and inflammatory mediators can induce thymic involution, characterized by reduced thymocyte numbers, cortical thinning, and stromal fibrosis. These pathological changes impair T-cell production, leading to immunosuppression and increased susceptibility to infections. Morphologically, the thymus may show lymphocyte depletion, increased apoptotic bodies, and disrupted architecture. Such alterations compromise the newborn's ability to mount effective immune responses, which is critical in the context of RDS where secondary infections are common. Understanding thymic pathology in RDS sheds light on the interplay between respiratory failure and immune dysfunction, highlighting the need for immune-supportive therapies.

The adrenal glands and thymus are functionally interconnected organs that coordinate the body's response to stress and immune regulation. In newborns with RDS, pathomorphological changes in these organs are often interdependent. Elevated glucocorticoid levels produced by the adrenal glands during stress can induce thymic atrophy by promoting apoptosis of thymocytes. Conversely, impaired thymic function reduces immune competence, potentially exacerbating systemic inflammation and adrenal stress responses. Histological examinations frequently reveal concurrent adrenal hyperplasia and thymic involution, indicating a coordinated adaptation to severe illness. This integrated response is essential for balancing inflammation and immune defense but may also contribute to immune suppression and vulnerability. Investigating these changes provides comprehensive understanding of the systemic effects of RDS and may guide combined endocrine and immunomodulatory treatment strategies.

Alterations in adrenal gland function and thymic integrity have profound clinical implications for newborns suffering from RDS. Increased adrenal activity helps the infant cope with physiological stress, maintain cardiovascular stability, and regulate inflammation. However, prolonged adrenal overactivation may lead to hormone imbalances and eventual exhaustion, complicating recovery. Meanwhile, thymic atrophy results in reduced immune surveillance and

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higher risks of secondary infections, which are major causes of morbidity and mortality in these patients. Monitoring adrenal and thymic changes can therefore assist clinicians in predicting disease severity, tailoring corticosteroid therapy, and implementing appropriate infection control measures. Early identification of these organ dysfunctions improves prognosis and can inform the development of novel supportive therapies targeting both endocrine and immune systems in RDS.

This study demonstrates that in newborns with Respiratory Distress Syndrome, the adrenal glands and thymus undergo significant pathomorphological alterations that are intricately linked. The adrenal glands respond to stress by increasing hormone production, while the thymus exhibits atrophic changes that impair immune function. These combined changes reflect the newborn's systemic adaptation to severe respiratory illness and highlight the importance of integrated organ responses in disease progression. Understanding this relationship is crucial for improving diagnostic accuracy and therapeutic approaches. Future research should focus on exploring treatments that support adrenal function and prevent thymic involution, thereby enhancing overall outcomes. Clinicians are encouraged to monitor endocrine and immune parameters closely in RDS patients to optimize care and reduce complications.

Discussion

The findings of this study highlight significant pathomorphological alterations in the adrenal glands and thymus of newborns affected by Respiratory Distress Syndrome (RDS). The observed adrenal cortical hyperplasia and hypertrophy indicate an intensified endocrine stress response, reflecting the organism's attempt to compensate for severe hypoxia and systemic imbalance. This adaptation is essential for maintaining homeostasis; however, prolonged adrenal stimulation may lead to functional exhaustion, negatively impacting the neonate's ability to manage stress and inflammation effectively. Simultaneously, thymic involution observed in RDS cases suggests a suppression of the immune system due to hypoxia and elevated glucocorticoid levels. The reduction in thymocyte populations and structural disruptions within the thymus compromise the development of competent T-cell-mediated immunity, thereby increasing vulnerability to infections. This immune suppression poses a critical risk, as secondary infections significantly contribute to morbidity and mortality in neonates with RDS.

The integral relationship between the adrenal glands and thymus underlines a complex neuroendocrine-immune interaction, where heightened cortisol production can induce thymic atrophy, creating a feedback loop that exacerbates immunodeficiency. This interconnected response is an important consideration for clinical management, suggesting that therapies targeting only respiratory function may overlook systemic organ dysfunction. Understanding these pathological changes reinforces the need for a holistic approach in treating RDS, incorporating endocrine and immunological support alongside respiratory care. Further research should investigate potential interventions aimed at preserving adrenal function and preventing thymic involution to improve overall neonatal outcomes.

Conclusion

In conclusion, Respiratory Distress Syndrome in newborns leads to notable pathomorphological changes in both the adrenal glands and the thymus, reflecting the systemic impact of this critical condition. The adrenal glands exhibit signs of hyperactivation as a response to physiological stress, which initially supports the neonate's adaptation but may result in eventual functional exhaustion. Concurrently, the thymus undergoes involution, impairing the newborn's immune capacity and increasing susceptibility to infections.

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The integral connection between these organs highlights the complex neuroendocrine-immune interactions during RDS, emphasizing that the syndrome affects more than just the respiratory system. These findings underscore the importance of comprehensive clinical management that addresses endocrine and immune dysfunctions alongside respiratory support. Future studies and therapeutic strategies should focus on protecting and restoring adrenal and thymic function to improve survival rates and long-term health outcomes in affected newborns.

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