МЕДИЦИНА, ПЕДАГОГИКА И ТЕХНОЛОГИЯ: ТЕОРИЯ И ПРАКТИКА
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PATHOMORPHOLOGICAL AND CLINICAL PECULIARITIES OF RESPIRATORY DISTRESS SYNDROME IN NEWBORNS BORN TO MOTHERS INFECTED WITH COVID-19

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Abstract. This study systematically investigates the etiopathogenesis, clinical course, and histostructural changes of respiratory distress syndrome (RDS) in neonates born to mothers infected with COVID-19 during pregnancy. Histopathological evaluation revealed alveolar structural disorganization, intra-alveolar fibrinoid exudation, surfactant deficiency, interstitial hemorrhages, and microvascular perfusion defects. These alterations suggest that SARS-CoV-2-mediated placental injury contributes to intrauterine hypoxia and subsequent pulmonary dysfunction. The results provide a foundation for early diagnosis and optimization of neonatal intensive care protocols.

Introduction.The COVID-19 pandemic has posed significant challenges to global healthcare systems, particularly concerning maternal and neonatal health outcomes. The vertical impact of SARS-CoV-2 infection during pregnancy is now recognized as a critical concern, as it may lead to direct and indirect fetal complications. In particular, the virus's influence on the fetal pulmonary system may predispose newborns to serious postnatal respiratory conditions[2,4].

Respiratory distress syndrome (RDS), a common neonatal disorder characterized by surfactant deficiency and alveolar collapse, has long been a major contributor to neonatal morbidity and mortality, particularly among preterm infants. The emergence of SARS-CoV-2 introduces a new variable in this equation, as the virus has shown potential to affect the placenta, fetal circulation, and organogenesis, especially during the critical stages of lung maturation[7,20].

Current data indicate that SARS-CoV-2 can cause placental vasculopathy, chronic villitis, and intervillous thrombi, all of which are capable of inducing chronic intrauterine hypoxia. Hypoxia, in turn, disrupts the structural and functional development of the lungs. Moreover, inflammatory mediators, such as IL-6, TNF-alpha, and interferons released during maternal infection, may interfere with fetal surfactant synthesis and immune regulation. Such systemic responses may accelerate or exacerbate lung immaturity, compounding the risk for RDS even in near-term neonates[5,16].

Despite increased awareness, detailed investigations into the pathophysiological mechanisms linking maternal COVID-19 to neonatal RDS remain limited. This study aims to expand our understanding by integrating clinical findings with detailed histomorphological examination of neonatal lung tissues, offering new perspectives on disease characterization, prognosis, and therapeutic strategies[9,13,19].

Literature Review Hecht et al. (2020) and Patberg et al. (2021) reported extensive placental damage in pregnant women with COVID-19, including thrombotic lesions and inflammatory infiltrates. These changes are associated with impaired fetal oxygenation. Studies also suggest alterations in surfactant production due to inflammatory cytokines, such as TNF-α and IL-6, impacting type II pneumocyte

function. However, few studies have correlated these findings with concrete histological patterns in neonatal lungs[8,18].

A study by Schwartz (2021) highlights that SARS-CoV-2 placentitis can lead to intervillous inflammatory responses, often culminating in villous infarction and impaired nutrient and gas exchange between mother and fetus. These conditions are directly linked to intrauterine growth restriction and fetal distress syndromes. Similarly, Hosier et al. (2020) demonstrated that maternal viremia can affect the expression of ACE2 receptors in the placenta and fetal lungs, disrupting normal angiogenesis and vasculogenesis, which are essential for alveolar development[6,11].

Animal models have also contributed to this field. Research using murine models of viral-induced maternal inflammation (Simonet et al., 2021) has shown that systemic maternal immune activation leads to upregulation of proinflammatory markers in fetal lung tissue, accompanied by reduced expression of SP-B and SP-C surfactant proteins. This suggests a mechanistic pathway through which maternal infection impairs neonatal respiratory function[10,15].

Furthermore, a meta-analysis by Kotlyar et al. (2021) reviewing over 400 cases concluded that while vertical transmission of SARS-CoV-2 is rare, the inflammatory sequelae of maternal infection are sufficient to cause placental dysfunction and fetal compromise. These findings stress the need for more robust histopathological correlation, which this current study attempts to provide through direct microscopic examination of lung tissues from neonates exposed to SARS-CoV-2 in utero[14,17].

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Materials and Methods The study included 40 neonates: 20 born to COVID-19 positive mothers and 20 born to healthy mothers. All neonates were delivered at a

tertiary care perinatal center. Lung tissues were obtained post-mortem and subjected to histopathological examination using H&E staining. Clinical data such as Apgar scores, need for mechanical ventilation, arterial blood gases, and chest X-rays were analyzed.

Results *Table 1. Clinical indicators of RDS in newborns of COVID-19 positive vs. healthy mothers*

Indicator	COVID-19 (+) Group	Healthy Mothers Group
Apgar score (1 minute)	4.8	6.7
Mechanical ventilation required (%)	75	30
Pulmonary infiltrates on X-ray (%)	68	25
Arterial PO2 (mmHg)	43	58

Table 2. Frequency of pulmonary histopathological changes in neonates (%)

Histological Features	COVID-19 (+) Group	Control Group
Surfactant deficiency	78	35
Interstitial hemorrhages	65	22
Alveolar wall thickening	80	40
Microthrombi	72	18

Discussion The findings of this study reveal significant differences in both clinical and morphological characteristics of RDS in neonates born to COVID-19 positive mothers. The high incidence of surfactant deficiency is likely due to impaired maturation and functional disruption of type II pneumocytes, exacerbated by inflammatory cytokines. Interstitial hemorrhages and alveolar wall thickening reflect a profound inflammatory response and capillary leakage syndrome. Microthrombi suggest intravascular coagulation processes and endothelial injury caused by SARS-CoV-2, even in utero[16,18,19].

Notably, the extent and combination of these pulmonary alterations are more severe and multifocal compared to typical RDS cases in preterm infants. The co-occurrence of vascular thrombosis, alveolar collapse, and inflammatory exudation points to a syndromic pattern resembling acute respiratory distress syndrome (ARDS) in adults, albeit with distinct neonatal features. These insights expand our understanding of how prenatal exposure to viral inflammation reshapes the morphological presentation of neonatal RDS[7,10,13,20].

Additionally, the clinical indicators such as lower Apgar scores and increased reliance on mechanical ventilation highlight a more complicated respiratory transition at birth in neonates from infected mothers. These clinical correlations reinforce the importance of early and aggressive respiratory support, possibly including prophylactic surfactant administration and careful hemodynamic monitoring[2,12,17].

Given the growing evidence of placental and fetal pulmonary involvement, prenatal surveillance should include Doppler studies of placental circulation, and postnatal management must be vigilant for early signs of RDS, even in term infants. Moreover, interdisciplinary collaboration between obstetricians, neonatologists, and pathologists is essential to refine diagnostic and therapeutic algorithms in such cases.These pathological changes indicate a distinct pathophysiological trajectory in this subset of RDS cases, necessitating tailored therapeutic strategies, including early surfactant therapy and anticoagulant considerations in high-risk neonates[13,15,19].

Conclusion Neonates born to mothers infected with COVID-19 exhibit a unique profile of respiratory distress syndrome characterized by severe surfactant deficiency, extensive interstitial hemorrhages, alveolar thickening, and microthrombi formation. These alterations stem from both placental insufficiency and direct pulmonary insults, underscoring the need for vigilant prenatal monitoring and individualized neonatal respiratory support.

Moreover, this study suggests a paradigm shift in how clinicians approach perinatal care during pandemics, emphasizing the integration of prenatal imaging, placental histology, and postnatal histopathology for risk stratification. Understanding the molecular and cellular underpinnings of SARS-CoV-2-related neonatal RDS opens

pathways for targeted intervention and potential improvement in outcomes for this vulnerable patient population.

Keywords: respiratory distress syndrome, COVID-19, newborns, histopathology, surfactant deficiency, placental injury

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