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MORPHOLOGICAL AND IMMUNOHISTOCHEMICAL CHARACTERISTICS OF LUNG TISSUE IN NEWBORNS BORN TO MOTHERS INFECTED WITH COVID-19


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Annotatsiya: Neonatal SARS-CoV-2 infeksiyasi kam uchrashi bilan birga, vertikal yoki perinatal yuqish holatlarida o‘pkada xarakterli morfologik va immunologik o‘zgarishlar namoyon bo‘lishi mumkin. Ushbu tadqiqotda COVID-19 musbat onalardan tug‘ilgan yangi tug‘ilgan chaqaloqlarning klinik ko‘rsatkichlari hamda autopsiya natijalari, gistopatologik, immunogistokimyoviy va o‘pka to‘qimalarining ultrastrukturaviy xususiyatlari asosida tahlil qilindi. Asosiy makroskopik va mikroskopik topilmalar qatorida diffuz alveolyar shikastlanish (DAL), II tip pnevmositlar giperplaziyasi, interstitsial mononuklear infiltratsiya, makrofaglarning to‘planishi va immun hujayra markerlarining (CD68, CD4, CD8, FOXP3, IL-6, IL-17) o‘zgaruvchan ifodasi qayd etildi. Shu bilan birga, joriy adabiyotdagi mavjud cheklovlar yoritilib, gistologik va immunogistokimyoviy o‘zgarishlarni aks ettiruvchi sxematik jadval taqdim etildi hamda kelgusida tizimli va keng qamrovli tadqiqotlar olib borish bo‘yicha tavsiyalar ishlab chiqildi.

Kalit so‘zlar: yangi tug‘ilgan chaqaloq, SARS-CoV-2, o‘pka patologiyasi, immunogistokimyo, alveolyar shikastlanish, II tip pnevmositlar, vertikal yuqish.

Аннотация: Неонатальная инфекция SARS CoV 2 встречается редко, но в случаях вертикальной или перинатальной передачи патология легких может отражать характерные морфологические и иммунные изменения. Мы рассматриваем имеющиеся клинические случаи и результаты вскрытий новорожденных, рожденных от матерей с положительным результатом на COVID 19, уделяя особое внимание гистопатологии, иммуногистохимии и ультраструктурным особенностям легочной ткани. Ключевые результаты включают диффузное альвеолярное повреждение (ДАП), гиперплазию пневмоцитов II типа, интерстициальную мононуклеарную инфильтрацию, накопление макрофагов и переменную экспрессию маркеров иммунных клеток (CD68, CD4, CD8, FOXP3, IL 6, IL 17). Мы

подчеркиваем ограничения в текущей литературе, предлагаем схематическую гистологическую/иммуногистохимическую таблицу и предлагаем направления для будущих систематических исследований.

Ключевые слова: новорожденный, SARS CoV 2, патология легких, иммуногистохимия, альвеолярное повреждение, пневмоциты II типа, вертикальная передача

Abstract: Neonatal infection with SARS-CoV-2 is rare, but in cases of vertical or perinatal transmission, lung pathology may reflect characteristic morphological and immune alterations. We review available case reports and autopsy studies of neonates born to COVID-19 positive mothers, focusing on histopathology, immunohistochemistry, and ultrastructural findings of pulmonary tissue. Key findings include diffuse alveolar damage (DAD), type II pneumocyte hyperplasia, interstitial mononuclear infiltration, macrophage accumulation, and variable expression of immune cell markers (CD68, CD4, CD8, FOXP3, IL-6, IL-17). We highlight limitations in the current literature, propose a schematic histologic/immunohistochemical table, and suggest directions for future systematic studies.

Keywords: Neonate, SARS-CoV-2, lung pathology, immunohistochemistry, alveolar damage, type II pneumocytes, vertical transmission

Introduction: The COVID-19 pandemic has raised urgent questions about vertical (in utero) or perinatal transmission of SARS-CoV-2 and its effects on the developing fetal and neonatal lung. While most newborns born to infected mothers are either uninfected or manifest mild disease, rare fatal neonatal cases provide unique windows into the pulmonary pathology associated with SARS-CoV-2 in early life. Because infant lung development differs substantially from adult lung architecture and immune milieu, a dedicated review and synthesis of neonatal lung pathology are warranted.

Adult autopsy studies in COVID-19 have documented patterns such as diffuse alveolar damage (both exudative and proliferative phases), hyaline membrane formation, alveolar septal thickening, microthrombi, endothelial injury, and fibroproliferation. However, the neonatal lung has distinct susceptibilities, surfactant biology, and immune responses. This manuscript reviews the available morphological and immunohistochemical evidence in neonates born to COVID-19 mothers, presents a summary table of expected findings, and outlines gaps for further research.

Methods: Because of the scarcity of large series, our approach is largely a narrative review supplemented by detailed case analyses. We systematically searched PubMed, Europe PMC, and Google Scholar using terms such as "neonate COVID-19 lung pathology," "neonate SARS-CoV-2 autopsy," "infant COVID-19 immunohistochemistry," and "vertical transmission SARS-CoV-2 lung." We included reports with histology, immunohistochemistry (IHC), and/or ultrastructural analysis of neonatal lung tissue. Where direct neonatal data were lacking, we cautiously reference adult COVID-19 lung pathology to suggest possible parallel mechanisms. We synthesized the findings into comparative morphological and immunohistochemical tables.

Results: Summary of Key Neonatal / Infant CasesBelow are selected cases with pulmonary pathology and immunohistochemical characterization:

1. Infant lethal COVID-19, 11-month oldIn a case of an 11-month infant who died of COVID-19 complications, lung histopathology revealed edema, hyaline membranes, airway mucus plugging, and interstitial inflammation. On the cellular level, there was a substantial increase in alveolar type II (ATII) pneumocytes (marked by cytokeratin 19, TTF-1, and napsin A). Transmission electron microscopy showed apoptotic features in ATII cells (chromatin clustering, nuclear condensation). IHC demonstrated a marked increase of $CD68^+$ macrophages in alveoli, elevated IL-6 in immune and stromal cells, moderate elevation of $FOXP3^+$ (T regulatory) and IL-17^+ cells, and expression of $CD4^+$ and $CD8^+$ T cells in alveolar walls.

2. Neonate autopsy (preterm, delivered ~25 weeks) in USAIn a neonate delivered at 25 weeks whose mother had asymptomatic SARS-CoV-2, autopsy showed severe diffuse alveolar damage (DAD) with type II pneumocyte hyperplasia, hyaline membranes, and interstitial mononuclear infiltrate. SARS-CoV-2 viral antigen was localized by immunohistochemistry in lung tissue. Additional IHC/in situ hybridization detected viral RNA in vascular endothelium (heart, liver).

3. Preterm newborn hyperinflammation case reportIn a preterm infant with fatal COVID-19, lung pathology showed severe bronchopneumonia with obliteration of airspaces by inflammatory cells. Immunohistochemistry of lung and heart demonstrated LDLR, NF-κB, OPN (osteopontin) and ICAM-1 expression, consistent with hyperinflammatory vascular injury. Also, citrullinated histone H3(citH3) expression implied neutrophil extracellular trap (NET) formation.

These limited reports converge on certain patterns, while many aspects remain unexplored (especially in term neonates or asymptomatic infections).

Figure 1. Pulmonary pathological features in coronavirus-associated severe acute respiratory syndrome: diffuse alveolar damage, hyaline membranes, alveolar edema and mononuclear infiltration observed in neonatal lung tissue.

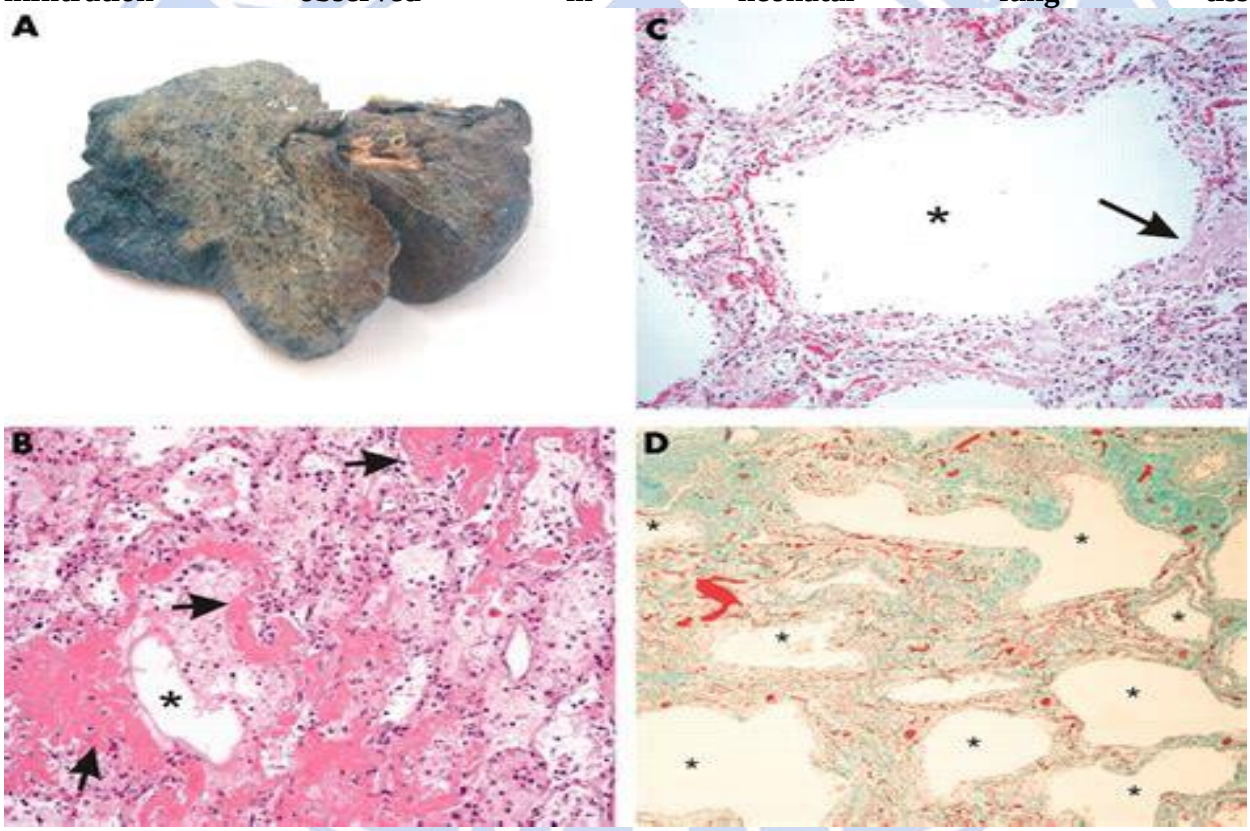
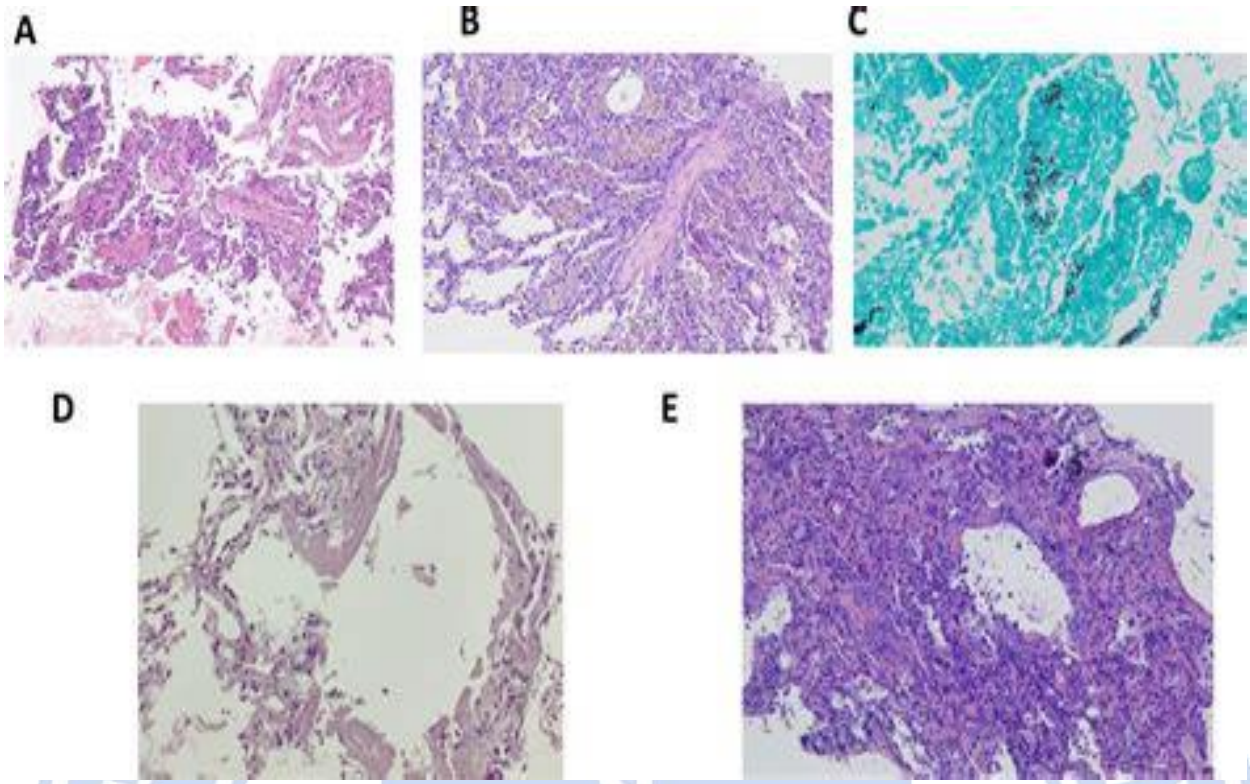


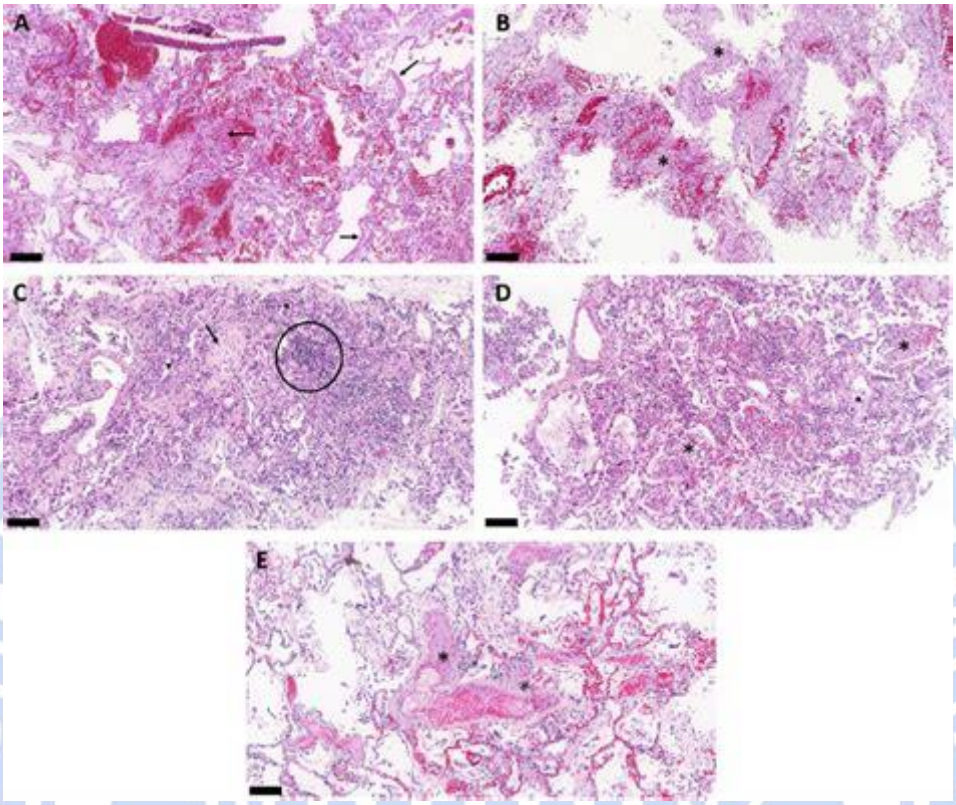
Figure 2. Histology Study of Postmortem Lung Biopsies in Patients With Covid-19 Demonstrates Diffuse Alveolar Damage and Interstitial Inflammation.



ImmunohistochemicalCellularImmuneFeatures

Marker / Cell Type	Findings in Neonatal / Infant Lungs	Interpretation / Notes
CD68⁺ macrophages	Markedly increased in alveolar spaces	Suggests robust innate macrophage response in neonatal lung
IL-6 (cytokine)	Elevated expression in immune and stromal cells	Reflects proinflammatory milieu, possible cytokine storm activation
CD4⁺ T lymphocytes	Moderate presence in alveolar walls	Adaptive immune infiltration present albeit moderate
CD8⁺ T lymphocytes	Present	Cytotoxic T cell presence in alveolar septa / walls
FOXP3⁺ T regulatorycells	Moderatelyincreased	Suggests attempts for immune regulation / balance
IL-17⁺ / Th17 cells	Elevatedmoderately	May contribute to inflammatory amplification
NF-κB, OPN, ICAM-1, LDLR (in preterm case)	Positive in vascular/inflammatory compartments	Indicates vascular activation, adhesion, immune signaling in neonatal lung injury
citrullinatedhistone H3 (citH3)	Positive	Marker of neutrophil extracellular trap (NET) formation, may exacerbate injury
Viral antigen / RNA (IHC / ISH)	In neonatal autopsy, SARS-CoV-2 spike protein staining in alveoli; viral RNA in lung and vascular endothelium	Confirms direct viral involvement in lung tissue in some neonatal cases

Figure 3. Diffuse alveolar damage patterns reflect the immunological and histopathological features of SARS-CoV-2 infection in neonatal lungs, including macrophage infiltration and T cell involvement.



Proposed Schematic Immunohistochemical Panel & Scoring (for future studies)

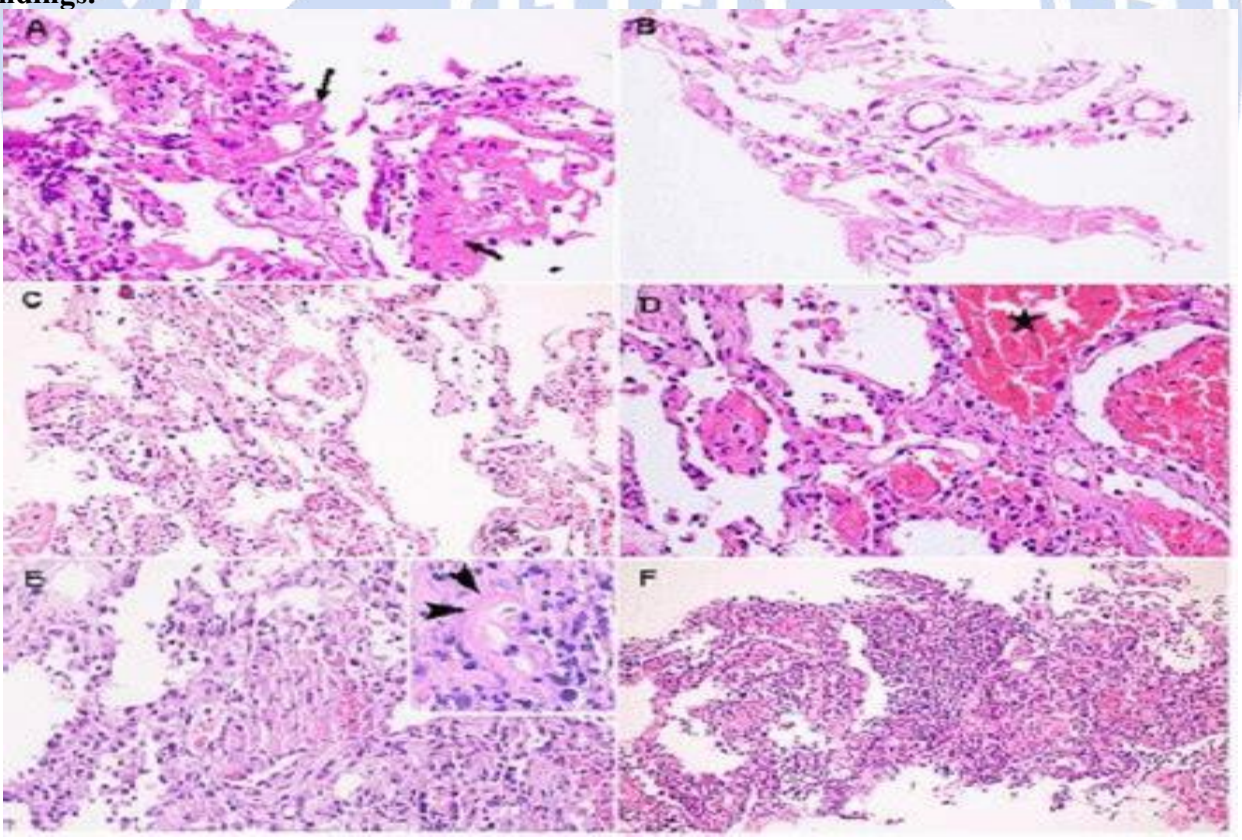
IHC Marker	Cell / Structure Target	Scoring (0–3+)	Rationale
Cytokeratin 19 / TTF-1 / Napsin A	ATII pneumocytes	0 = absent, 1 = focal, 2 = moderate, 3 = marked hyperplasia	To quantify pneumocyte hyperplasia / proliferation
Caspase-3 / cleavedcaspase	Apoptoticpneumocytes	0–3+	To assess degree of apoptosis in alveolar epithelium
CD68	Alveolar/macrophages	0–3+	Assessmacrophageinfiltration
CD163 / CD206	M2-type macrophages	0–3+	To distinguishmacrophagepolarization
CD4, CD8	T lymphocytes	0–3+	Evaluate adaptive T cell infiltration
FOXP3	T regulatorycells	0–3+	Gaugeimmuneregulatorycounterbalance
IL-6, IL-1β, TNFα	Cytokineexpressionintissue	0–3+	Measureproinflammatorycytokineburden
IL-17	Th17 responses	0–3+	To assess contribution of Th17 pathway

IHC Marker	Cell / Structure Target	Scoring (0–3+)	Rationale
NF-κB (p65)	Nuclear activation in immune and stromal cells	0–3+	Marker of inflammation signaling activation
ICAM-1, VCAM-1	Endothelial / vascularactivation	0–3+	To detect vascular involvement / adhesion molecule upregulation
Citrullinatedhistone H3 (citH3)	NET formation	0–3+	To detect neutrophil extracellular traps
SARS-CoV-2 spike or nucleocapsid protein (IHC) & in situ hybridization (ISH)	Viralantigen / RNA	positive / negativeorgraded	To localize viral presence in lung tissue

Table 1. Proposed IHC Panel for Neonatal Lung SARS-CoV-2 Pathology

In actual studies, this panel could be supplemented by immunofluorescence (double staining), multiplex IHC, or spatial transcriptomics to delineate cell–cell interactions.

Figure 3. Histopathological observations in COVID-19: a systematic review shows hyaline membranes, interstitial infiltration, and epithelial injury consistent with neonatal lung findings.



DiscussionInterpretation of Findings The presence of DAD with hyaline membranes and edema in neonates suggests that severe SARS-CoV-2 infection can produce classic adult-type alveolar

injury even in early life, although the incidence is undoubtedly lower.ATII pneumocyte hyperplasia may represent both a reparative response and a viral target, given that SARS-CoV-2 preferentially infects alveolar epithelial cells. In the infant case, ultrastructural evidence of apoptosis in ATII cells supports the hypothesis of direct injury.

Macrophage accumulation ($CD68^+$) appears robust, indicating that innate immune activation is a major component even in neonatal lungs.Moderate infiltration by $CD4^+$, $CD8^+$, $FOXP3^+$, and $IL\text{-}17^+$ T lymphocytes shows that the adaptive arm may also be engaged, though perhaps not with the same intensity as adult viral pneumonia.Elevated IL-6 expression and activated NF-κB / adhesion molecules in some cases reflect a hyperinflammatory / cytokine cascade contribution, possibly exacerbating alveolar injury and vascular damage.The presence of NET markers (citH3) in neonatal lung is particularly intriguing, suggesting that neutrophil-mediated microvascular / alveolar injury could also participate.

Challenges, Limitations, andGaps

1. **Extremely limited sample size** — Only a handful of neonatal / infant lung pathology cases exist, making it difficult to draw generalizable conclusions.
2. **Bias toward severe/fatal cases** — All published neonatal lungs with histology come from fatal or near-fatal infections; the spectrum in milder or asymptomatic neonates remains unknown.
3. **Lack of term neonate data** — Most cases are preterm, infants, or older; direct data on term newborns infected in utero are minimal.
4. **Postmortem artifact and sampling constraints** — Autolysis, postmortem changes, and limited sampling may obscure subtle features.
5. **Absence of longitudinal or comparative (control) cohorts** — Without matched control neonatal lungs (e.g. noninfected) or systematic sampling, quantification is difficult.
6. **Limited multiplex / advanced methods** — Spatial transcriptomics, single-cell RNA sequencing, or multiplexed IHC would better delineate cell–cell interactions, but are rarely applied in these rare specimens.
7. **Uncertainty in timing of infection** — It is often unclear if infection was prenatal, perinatal, or postnatal, complicating interpretation of whether lesions are developmental, prenatal, or reactive.

Proposed Model / Hypothesis for Neonatal SARS-CoV-2 Lung Injury Direct viral injury to alveolar epithelium (especially ATII pneumocytes) leads to apoptosis, denudation, and triggers a reparative hyperplasia.

Innate immune activation, predominantly macrophages, senses tissue injury and viral antigen, producing cytokines (IL-6, IL-1β, TNFα) that amplify inflammation.

Adaptive immune recruitment (CD4, CD8, FOXP3, IL-17) may contribute to local regulation or further injury, depending on the balance.

Vascular / endothelial involvement — upregulation of adhesion molecules and endothelial activation could promote microvascular permeability, contributing to edema and interstitial injury.

NET-mediated damage in severe cases may exacerbate microvascular injury and alveolar damage.Because neonatal lungs are ongoing in alveolarization and have different immunologic tolerance, the balance between injury and repair may differ from adults — possibly making neonates more vulnerable to dysregulated inflammation in severe cases.

Conclusion & Future Directions Neonatal lung pathology in the context of maternal SARS-CoV-2 infection, though rarely documented, reveals that even in early life, COVID-19 can induce hallmark features of diffuse alveolar damage, pneumocyte hyperplasia, and immune cell infiltration. Key immunohistochemical markers (CD68, CD4, CD8, FOXP3, IL-6, citH3) are detectable and provide insight into the interplay between viral injury and innate/adaptive immunity.

To move the field forward, we recommend:Establishing multicenter neonatal /perinatal pathology registries to collect more cases, especially in nonfatal and asymptomatic neonates.Applying standardized IHC panels (as sketched above) and digital morphometry for interstudy comparability.Using multiplex immunofluorescence, spatial transcriptomics, or single-cell sequencing on neonatal lung tissue to map cell interactions, cytokine microenvironments, and viral localization.Performing comparative controls with age-matched noninfected neonatal lungs to distinguish COVID-19–specific changes from baseline developmental variation.Investigating longitudinal outcomes, where feasible, by correlating pathology (or surrogate biomarkers) with clinical respiratory trajectories in survivors.

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