American Journal of Medicine and Medical Sciences 2025, 15(9): 3010-3014
DOI: 10.5923/j.ajmms.20251509.35

Distinctive Pathomorphological and Morphometric Features of Various Forms of Pneumopathies

Abdullaeva Nurjaxon Juraboy qizi[1,*], Mamataliev Avazbek Ruziyevich[2]

[1]Assistant, Andijan State Medical Institute, Andijan, Uzbekistan
[2]PhD, Andijan State Medical Institute, Andijan, Uzbekistan

Abstract This study investigated the dynamics of pathomorphological and morphometric changes, as well as dysfunctional alterations, in the development of various clinical and morphological manifestations of pneumopathies in fetuses and newborns of different gestational ages. The research utilized forensic histological and pathohistological archival materials (107 cases) of lung tissues obtained from autopsies of stillborns and infants who died during the intranatal, early, and late neonatal periods in maternity complexes and perinatal centers of the Andijan region between 2021 and 2024. The frequency, prevalence, and severity of pulmonary pathomorphological and morphometric changes in pneumopathies generally depended on the gestational age of the newborn, prematurity or maturity, and the degree of lung tissue differentiation. Hemodynamic disturbances in the lungs were found to cause damage to alveolocytes and alveolar capillaries, disruption of the alveolo-capillary (air-blood) barrier, and transudation of serum proteins, thereby blocking the respiratory surface of the lung parenchyma.

Keywords Gestational age, Fetus, Newborn, Pneumopathy, Pathomorphology, Morphometry, Atelectasis, Hyaline membrane, Edema, Hemorrhage, Acute respiratory distress syndrome

1. Introduction

According to the World Health Organization, respiratory system diseases represent one of the most common pathologies and leading causes of mortality among newborns [5,6,7]. Respiratory distress syndrome (RDS), acute respiratory distress syndrome (ARDS), and other pneumopathies are reported to occur at a frequency ranging from 1.5 to 78.9 cases per 100,000 populations in different countries. UNICEF data (2017) indicate that in Central Asian countries, ARDS is among the primary causes of stillbirth and neonatal mortality [1,2].

These neonatal pathologies are considered non-inflammatory processes of the lungs and are manifested by atelectasis, edema, hemorrhagic changes, hyaline membranes, and related morphologic features [3,4]. Therefore, investigating the pathomorphological and morphometric characteristics of pneumopathies in neonates, clarifying their pathogenetic mechanisms, and assessing their clinical manifestations remain highly relevant scientific and practical issues in perinatal medicine and neonatology [1–7]. Recent epidemiological data report that RDS accounts for 26% of all neonatal ICU admissions in Egypt, with hyaline membrane disease and pneumonia being the leading causes of neonatal mortality in this population [8]. Globally, over 40% of all childhood deaths occur in the neonatal period, especially among infants born before 32 weeks of gestation, highlighting prematurity as a critical driver of respiratory mortality [9]. In 2023, the incidence of neonatal ARDS (NARDS) was estimated at 1.44%, with pneumonia, perinatal asphyxia, and early-onset sepsis among the top etiologies [10]. Advances in imaging and machine learning now support the classification of neonatal lung pathologies—such as RDS, TTN, and pneumonia-via automated analysis of lung ultrasound images, offering promise for rapid diagnosis in resource-limited settings [11]. Moreover, in new forms of bronchopulmonary dysplasia (BPD), current histopathological understanding reveals alveolar simplification, reduced septation, and persistent hyaline membranes indicating long-term structural impairment [12].

* Corresponding author:
abdullaevalucmira@gmail.com (Abdullaeva Nurjaxon Juraboy qizi)
Received: Aug. 25, 2025; Accepted: Sep. 17, 2025; Published: Sep. 23, 2025
Published online at http://journal.sapub.org/ajmms

2. Purpose of the Study

To investigate the distinctive pathomorphological and morphometric features of various forms of pneumopathies.

3. Materials and Methods

The objects of the study consisted of archival materials of

107 cases of lung tissue samples obtained during autopsies of fetuses and newborns who died from pneumopathies in the perinatal period at maternity complexes of the Andijan region between 2020 and 2024. The study materials included cases of different gestational ages and perinatal periods. Pathomorphological and morphometric parameters were examined in accordance with gestational and neonatal age. The obtained data were analyzed using statistical methods.

4. Results and Discussion

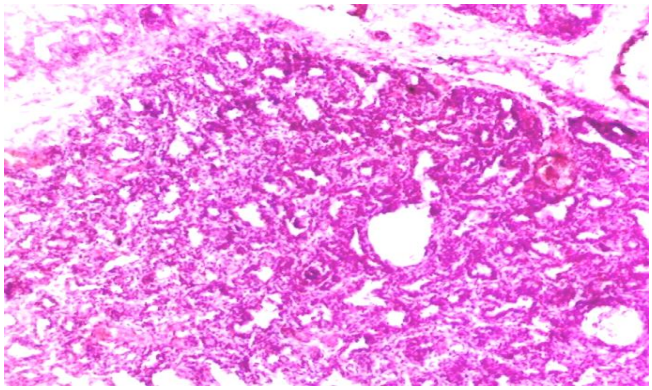

Figure 1. In immature lung tissue, respiratory bronchioles and some alveoli are partially opened, irregular in shape; stromal edema is present along with foci of primary atelectasis; alveocytes are not yet developed; moderate venous congestion and focal diapedetic hemorrhages are observed. Hematoxylin–eosin staining. Magnification ×4 ×12.5

It is of great importance to accurately identify the specific pathomorphological and morphometric features of alveolar structures in lung tissue affected by pneumopathies in fetuses of different gestational ages and in newborns. In fetuses at 22–28 weeks of gestation, macroscopic and microscopic examinations revealed immaturity of the lungs, characterized by total atelectasis, failure of alveoli to expand, or the presence of only narrow slit-like openings. The respiratory epithelium of the alveolar compartments appeared flattened; arterioles were short; interalveolar connective tissue septa were wide; type I alveocytes were more frequently damaged compared to type II alveocytes; and cellular lysis occurred more rapidly (Figure 1).

Between 29–31 weeks of gestation, an increase in the branching of terminal acini was observed, along with the differentiation of type I alveocytes, maturation of type II alveocytes, surfactant production, thinning of the mesenchyme, and the approximation of capillaries to type I alveocytes. Fetal lung fluid production was noted. Moderate venous congestion, primary atelectasis, diapedetic hemorrhages, and desquamation of the epithelial lining of the bronchioles were also observed. During this period, the atelectatic and edematous-hemorrhagic forms of pneumopathies predominated.

At 32–37 weeks of gestation, the formation of acini was completed, with differentiation between terminal and respiratory sacs. The number and depth of alveoli gradually increased, while the development of the capillary network in the interalveolar septa and flattening of the epithelium continued. Both small and large foci of atelectasis, distelectasis, focal hyaline membranes, diapedetic hemorrhages, venous congestion, serous edema, and desquamation of the bronchiolar epithelial lining were detected. During this period, the atelectatic and hyaline membrane forms of pneumopathies were predominant.

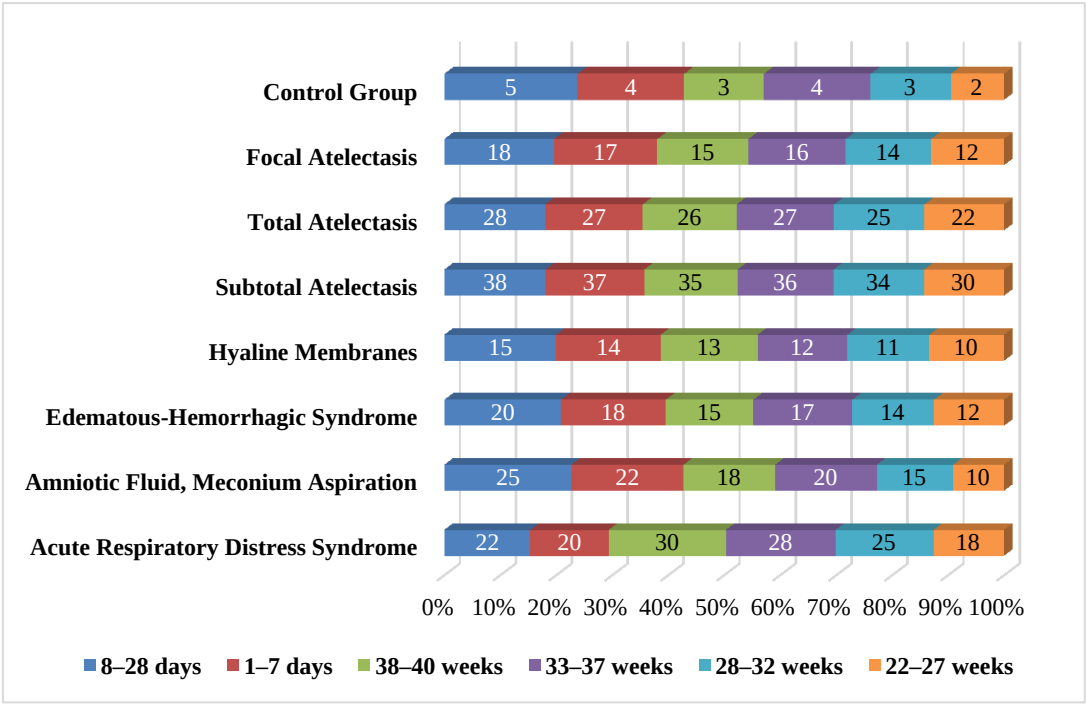

Figure 2. Changes in the diameters of type I alveocytes (µm) in various forms of pneumopathies

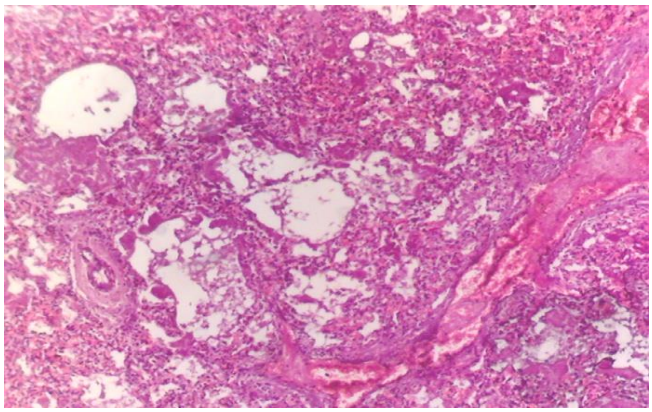

Figure 3. Partially opened bronchioles and alveolar spaces containing desquamated epithelium and amniotic fluid; initial formation of alveoli; moderate venous congestion; primary atelectasis; diapedetic hemorrhages; stromal edema. Hematoxylin–Eosin staining. Magnification 4×12.5

In stillborn fetuses of 32–37 weeks, as compared to those who survived briefly after birth, small and large foci of atelectasis, distelectasis, focal hyaline membranes, diapedetic hemorrhages, and a small number of leukocytes were recorded (Figure 2 and 3).

At 38–40 weeks of gestation, venous congestion, multiple small foci of primary atelectasis, hyaline membranes, diapedetic hemorrhages, serous edema, and desquamation of the epithelial lining of the bronchiolar mucosa were observed. Macroscopically, areas of atelectasis appeared meaty, reduced in size, and compact, with a grayish coloration compared to the surrounding tissues, manifesting as small focal lesions. During this period, the atelectatic and edematous-hemorrhagic forms of pneumopathies were found to predominate (Figure 4).

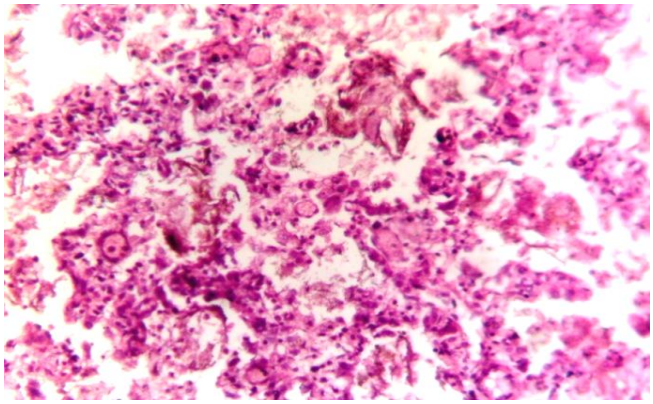

Figure 4. Formation of respiratory bronchioles and acini; alveoli are open, varying in size and shape; the number of type I alveocytes is increased, appearing hyperchromatic, round, and oval; type II alveocytes are fewer in number, larger in size, and hyperchromatic. Hematoxylin–eosin staining. Magnification A–B, 10*12.5

In newborns during the first 1–7 days of life, venous congestion in internal organs and hemodynamic disturbances in the microcirculatory bed were observed, accompanied by primary and secondary atelectasis in the lung tissue, desquamation of the bronchiolar mucosal epithelium, diapedetic hemorrhages, and serous edema. Hyaline membranes and constrictive arteritis in small arteries were also noted (Figure 5 and 6).

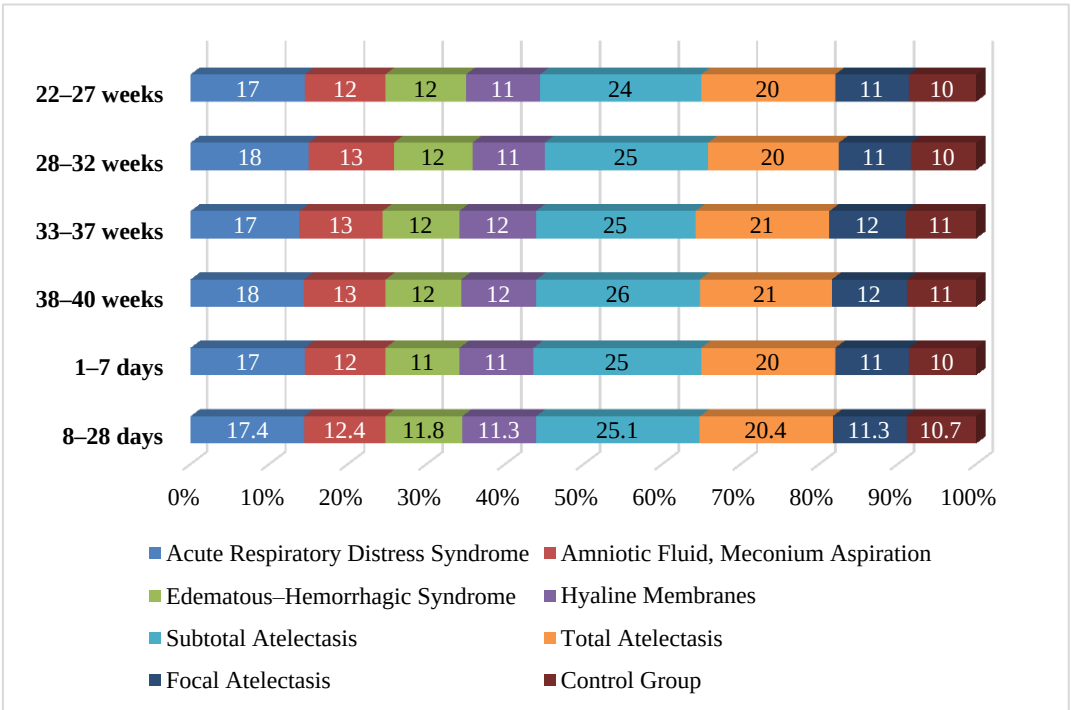

Figure 5. Changes in the diameters of type II alveocytes (μm) in various forms of pneumopathies

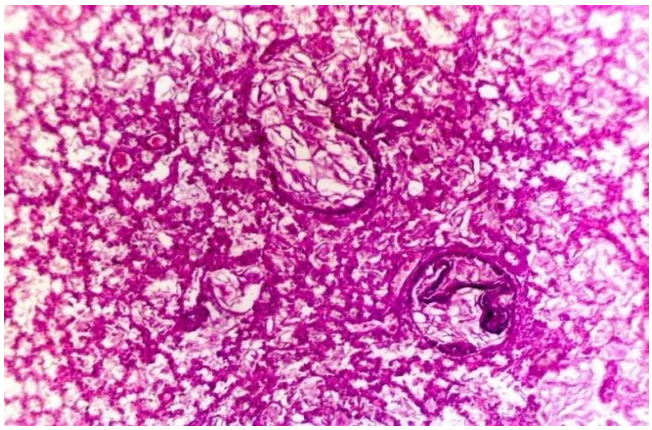

Figure 6. Respiratory bronchioles and alveoli are open; their lumens contain amniotic fluid and keratinized epithelium. Desquamation of the bronchiolar, alveolar epithelium, and alveocytes is observed, along with moderate venous congestion and interstitial edema. Hematoxylin–eosin staining. Magnification 10*12.5

During days 8–28 of life, the respiratory tract was fully formed, and the maturation of surfactant occurred in parallel with the differentiation and vascularization of certain respiratory segments. In the lungs, venous congestion, edema, and diapedetic hemorrhages were more frequently observed, while atelectasis and emphysema were not detected. The edematous–hemorrhagic form of pneumopathy was found to be predominant. The development of focal pneumonia was noted as a result of alterative and proliferative processes (Figure 7 and 8).

Micropreparations were scanned using the NanoZoomer system with NDP.view 2 software, and the resulting images were subjected to automated statistical processing through QuPath-0.5.0. In the analysis, the total area of all structural units was considered as Vv (100%), and the area of each specific structure was calculated and designated according to its name.

Morphometric parameters included type I alveocytes (a1), type II alveocytes (a2), stromal thickness (s), terminal airway diameter (t), hyaline membranes (gm), and the alveolar luminal activity coefficient (ABAC).

The morphometric study demonstrated that, in various forms of pneumopathies and with the progression of gestational age and perinatal periods, the diameter of type I alveocytes increased by 4–5 times compared with the control group, while type II alveocytes increased by 2–2.5 times. The thickness of the alveolar stroma was reduced approximately twofold, the diameter of the terminal airways expanded 2–3 times, and the hyaline membranes thickened by 1–1.5 times. These morphometric alterations reflect the distinct pathological changes characteristic of the different forms of pneumopathies.

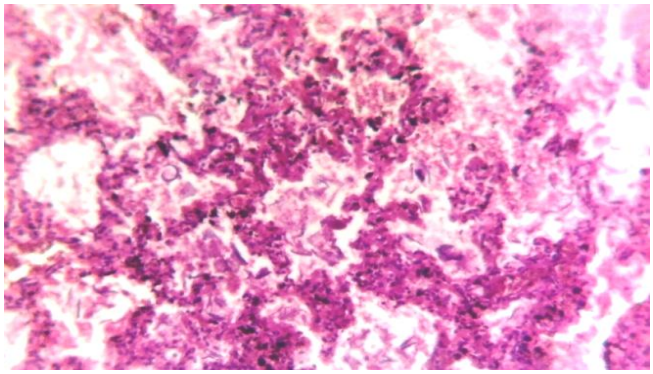

Figure 7. Desquamation of the mucosal lining of the respiratory bronchioles and alveoli; presence of serous–hemorrhagic fluid within the lumen; thickening of the interalveolar septa; edema; and infiltration of the stroma with lymphoid cells. Hematoxylin–Eosin staining. Magnification 10×12.5

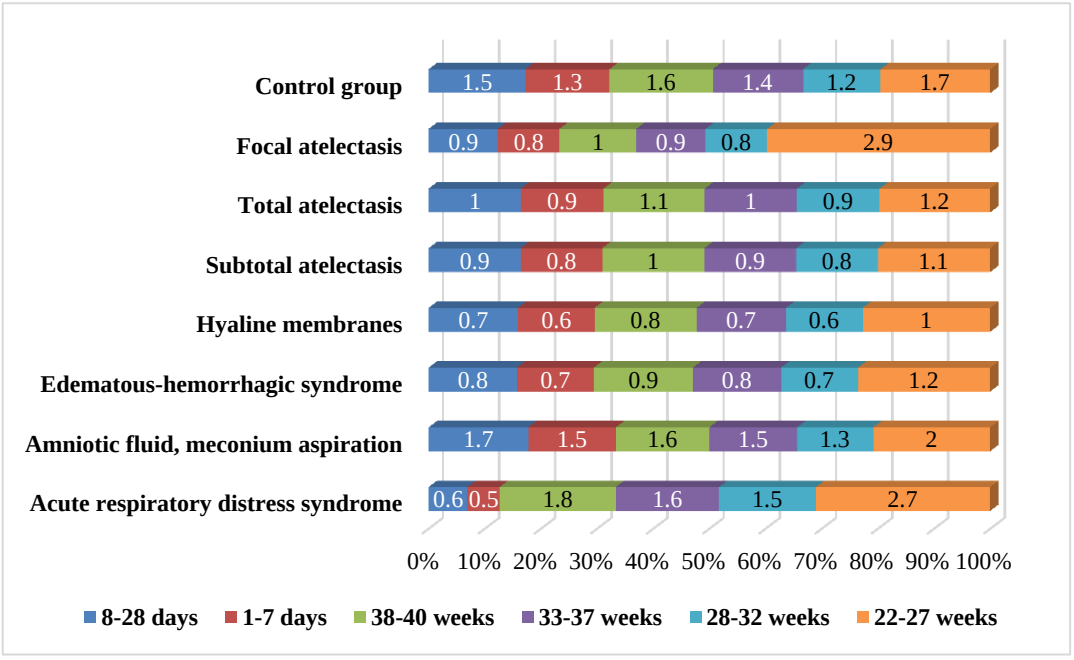

Figure 8. Changes in the alveolar lumen activity coefficient in various forms of pneumopathies

5. Conclusions

The frequency, distribution, and severity of pathomorphological and morphometric alterations in pneumopathies are strongly dependent on the gestational age of the newborn, the degree of prematurity or maturity, and the level of differentiation of lung tissue. The findings demonstrate that hemodynamic disturbances within the lungs contribute to significant injury of alveocytes and alveolar capillaries, resulting in disruption of the integrity of the alveolo-capillary barrier. This, in turn, promotes transudation of serum proteins, particularly fibrinogen, into the alveolar lumen, which blocks the respiratory surface and impairs gas exchange.

Moreover, morphometric analysis revealed that in pneumopathies of varying forms, type I alveocytes undergo a disproportionate enlargement compared to controls, while type II alveocytes also increase but to a lesser extent. Concurrently, thinning of the alveolar stroma and dilation of terminal airways reflect compensatory remodeling processes, whereas thickening of hyaline membranes signifies irreversible damage and altered barrier function.

These structural and functional alterations collectively indicate that the pathogenesis of pneumopathies is multifactorial, involving immaturity of lung tissue, surfactant deficiency, circulatory disturbances, and inflammatory–proliferative reactions. In premature infants, the predominance of atelectatic, hyaline membrane, and edematous–hemorrhagic forms underscore the critical role of lung immaturity and surfactant insufficiency, while in later gestational stages, compensatory processes and inflammatory responses contribute more prominently to the disease pattern.

Therefore, the study emphasizes that the pathomorphological and morphometric features of pneumopathies not only provide insight into the underlying mechanisms of neonatal respiratory disorders but also highlight their clinical significance. Understanding these changes is essential for improving diagnostic accuracy, guiding therapeutic interventions, and developing preventive strategies to reduce neonatal morbidity and mortality associated with pneumopathies.

REFERENCES

[1] Bokonbaeva, S. D., et al. (2024). Etiology of respiratory distress syndrome in premature newborns in Bishkek, Kyrgyz Republic. *Mezhdunarodnyy zhurnal prikladnykh i fundamentalnykh issledovaniy* [International Journal of Applied and Fundamental Research], (8), 9–12. (in Russian)

[2] Jumanov, Z. E., et al. (2022). Aspects of morphological changes in the cranial structures of newborns who died from the atelectatic form of pneumopathy. *Vestnik Vracha* [Journal Vestnik Vracha], 137–140. (in Uzbek/Russian)

[3] Dilmurodova, K. R., et al. (2021). *Muddatida tug'ilgan chaqoloqlar* [Full-term newborns: A study guide]. Samarqand: Tibbiyotko‘zgusi. (in Uzbek)

[4] Shabalov, N. P. (2023). *Neonatology: A textbook* (Vol. 1). Moscow: GEOTAR-Media. (in Russian)

[5] Moller, A. B., et al. (2017). Early antenatal care visit: A systematic analysis of regional and global levels and trends of coverage from 1990 to 2013. *The Lancet Global Health*, 5(10), e977–e983.

[6] Patel, R. M. (2016). Short- and long-term outcomes for extremely preterm infants. *American Journal of Perinatology*, 33(3), 318–328.

[7] Xu, J., et al. (2018). Deaths: Final data for 2016. *National Vital Statistics Reports*, 67(5), 1–76.

[8] Baseer, K. A. A., Mohamed, M., & Abd-Elmawgood, E. A. (2020). Risk factors of respiratory diseases among neonates in Neonatal Intensive Care Unit of Qena University Hospital, Egypt. *Annals of Global Health*, 86(1), 22–28..

[9] Ekhaguere, O. A. (2022). Respiratory distress syndrome management in resource-rich and resource-limited settings: implications of neonatal mortality. *Frontiers in Pediatrics*, 963–970.

[10] Chen, L., et al. (2023). Clinical characteristics and outcomes in neonates with ARDS. *eClinicalMedicine*, 101–110.

[11] Aujla, S., et al. (2023). Classification of lung pathologies in neonates using dual-tree complex wavelet transform. *Biomedical Engineering Online*, 45–56.

[12] Dankhara, N., et al. (2023). Bronchopulmonary dysplasia: pathogenesis and histopathology in the modern era. *Journal of Clinical Medicine*, 5001–5015.