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A review of clinical diagnosis and treatment of bronchopulmonary dysplasia in neonates

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ABSTRACT
Bronchopulmonary dysplasia is a chronic lung disease that predominantly affects newborns, particularly those born extremely early or very prematurely. The risk of developing this condition is higher in these infants. Under the influence of various factors, the pathological process is highly complex and significantly impacts the quality of life for newborns. This study analyzes the general situation of bronchopulmonary dysplasia in newborns and explores diagnostic and therapeutic measures for this condition.

KEYWORDS
neonate; bronchopulmonary dysplasia; clinical diagnosis and treatment

Bronchopulmonary dysplasia primarily results from reduced surfactant production in the lungs and incomplete development of the pulmonary vascular system, leading to abnormal breathing in infants. In recent years, with the advancement of assisted reproductive technologies, more premature babies have been born, significantly increasing the incidence of bronchopulmonary dysplasia. Statistics show that the incidence rate of bronchopulmonary dysplasia at 25-27 weeks gestation is as high as 45.0%. If not promptly controlled, infants may develop bronchitis and lung infections, requiring prolonged hospital stays and placing a significant financial burden on families. Additionally, it poses a substantial threat to the safety of the infant's life[1]. There is no definitive clinical consensus on neonatal bronchopulmonary dysplasia; factors such as genetics, inflammation, barotrauma, infection, and hypoxia can all contribute to lung damage and developmental issues[2]. Currently, there are no specific treatment options available for bronchopulmonary dysplasia in clinical practice.

1 Overview of neonatal bronchopulmonary dysplasia

As early as 1967, clinical descriptions of bronchopulmonary dysplasia have been described, in which children are mainly caused by lung injury during oxygen inhalation and mechanical ventilation, which is also called "old" bronchopulmonary dysplasia or classic bronchopulmonary dysplasia.

Research by [3] et al. found that it is more common in premature infants, with an average gestational age below 34 weeks and a birth weight under 2.2 kg. After severe respiratory distress syndrome, the lung surface lacks active substances, leading to chronic inflammation in the lung parenchyma, fibrotic changes, hyperplasia of airway smooth muscle, and squamous metaplasia. Affected by the condition, the lower respiratory tract is prone to recurrent infections, and the risk of secondary pulmonary hypertension and airway hyperresponsiveness increases, making feeding difficult and affecting normal growth and development of newborns. In June 2000, the National Institutes of Health (NICHD/NHLBI/ORD) redefined bronchopulmonary dysplasia based on epidemiology, etiology, and prognosis, indicating the need for prolonged mechanical ventilation and a high dependence on oxygen therapy during later recovery, with continuous oxygen use lasting over 28 days. For infants over 32 weeks of gestation, corrected to 36 weeks, the condition can be classified based on oxygen use at discharge: no oxygen use is categorized as mild, oxygen concentration below 30% as moderate, and oxygen concentration above 30% requiring mechanical ventilation as severe. If the fetus reaches 32 weeks or more, the condition is classified based on 56 days after birth or reference to the oxygen concentration at discharge, observing a significant reduction in the number of alveoli, larger volume, and a single structure, with mild fibrotic changes in the airways.

2 Etiology of bronchopulmonary dysplasia in newborns

The etiology of bronchopulmonary dysplasia in newborns is very complex. Children are often affected by multiple factors such as pressure injury, oxygen poisoning, heredity, volume injury, infection and inflammation, so that the lungs are not mature, resulting in lung injury and abnormal repair of lung tissue.

2.1 Lung maturity

Prenatal infants under 28 weeks of gestation have not yet fully developed their lungs, and they are at a critical stage of transitioning from the vesicular phase to the alveolar phase. Tian Li et al.[4] found that high volume fraction oxygen exposure can easily lead to barotrauma and volume injury. Moreover, under the influence of inflammation and other factors, it is more likely to result in delayed lung development, leading to bronchopulmonary dysplasia.

2.2 Infection and inflammatory injury

Al-JEBAWI et al. in the [5] study found that intrauterine perinatal infection is a significant cause of preterm birth and bronchopulmonary dysplasia. Moreover, postnatal infections and prenatal chorioamnionitis can lead to inflammatory responses, which also cause damage to the lungs, resulting in bronchopulmonary dysplasia. Therefore, in research on bronchopulmonary dysplasia, it is crucial to pay attention to inflammatory factors and infection responses.

2.3 Oxygen poisoning

Exposure of lung tissue to high oxygen partial pressure leads to inflammatory damage, edema, and fibrin deposition. Moreover, under the influence of highly reactive oxygen free radicals, cellular metabolism is impaired, causing structural damage and resulting in pulmonary injury. Research by Zhao Yipin et al. [6] found that under the effect of high oxygen partial pressure, a large amount of inflammatory mediators are released. Under the influence of chemotaxis, capillaries become more permeable, pulmonary vessels constrict, fibroblasts proliferate upon stimulation, and fibrin secretion increases, leading to pulmonary fibrosis. Premature infants, due to their immature antioxidant enzyme systems, have a higher risk of oxygen toxicity, which can cause damage to lung tissue.

2.4 Pulmonary interstitial edema

In a study by Yu Xiaoyan et al. [7], it was found that excessive sodium supplementation and infusion during treatment of premature infants can easily cause interstitial edema of lung, which is also an important cause of bronchopulmonary dysplasia. Therefore, attention should be paid to water and sodium intake control in the prevention of bronchopulmonary dysplasia.

2.5 Mechanical ventilation injury

The trachea and bronchial tree are structurally damaged under the influence of pressure injury, volume injury, etc. LANA MUKHARESH et al. [8] found that PS deficiency, affected by the cascade reaction of inflammatory factors, damages the epithelium of pulmonary bronchioles, leading to alveolar collapse, which affects the development of alveoli in children and results in a decrease in the number of alveoli.

3 Diagnosis of bronchopulmonary dysplasia in newborns

Studies show that the incidence of bronchopulmonary dysplasia varies both domestically and internationally due to differences in living conditions and diagnostic criteria. Currently, imaging techniques are more commonly used for diagnosing bronchopulmonary dysplasia in newborns. Liu Yibo et al. [9] found that in chest X-ray diagnosis of bronchopulmonary dysplasia in newborns, the lung fields show obvious ground-glass changes, with uneven density and linear or patchy shadows, and small cystic cavities are also observed. As the condition worsens, the translucent areas in both lung fields become significantly enlarged with vesicular changes, featuring linear or patchy shadows. Both lungs exhibit atelectasis and overinflation, as well as structural disarray. Some newborns show no typical findings on X-ray examination; the lung texture contours appear blurred on chest films, with overinflation, increasing the clinical diagnostic difficulty. Chest CT has higher image resolution, revealing symmetrical lesions in both lower lungs, but its low sensitivity can affect the doctor's diagnosis.

In recent years, modern medical technology has made significant progress, and hematological indicators can provide more diagnostic evidence for bronchopulmonary dysplasia in newborns. Li Guiju et al. [10] found that interleukin-3 (Interleukin-3, IL-3) and other inflammatory markers of the interleukin family play a crucial role in bronchopulmonary dysplasia. These inflammatory markers bind to receptors, activating mast cells and disrupting the balance of the mucosal environment, leading to inflammation and immune responses in the mucosal tissues. Exosomes are rich in cholesterol and sphingomyelin substances on their surface, and cells use mRNA, microRNA, and protein cellular products for precise delivery. In receptor-mediated interactions, membrane receptors and various biologically active molecules are stimulated

to transfer, exerting biological effects on target cells.

4 Treatment of bronchopulmonary dysplasia in newborns

4.1 Nutritional support

Providing energy and protein to children through nutritional support can effectively enhance the body's ability to fight infections, further reducing lung inflammation damage. [11] studies by MOHAB, GHANEM, et al. have shown that for children with bronchopulmonary dysplasia, it is important to supplement long-chain polyunsaturated fatty acids and glutamine in nutritional support to promote rapid growth and repair of lung tissue.

4.2 Mechanical ventilation

Bronchopulmonary dysplasia can achieve good therapeutic effects through mechanical ventilation, which primarily aims to maintain hemodynamic stability and reduce the risk of complications in patients. However, high-frequency ventilators often lead to hypoxia and low pressure during mechanical ventilation, causing a struggle between the patient and the machine. Chen Zhaoqiu et al. [12] found that compared to conventional assisted ventilation, high-frequency oscillatory ventilation can achieve a frequency more than four times higher. With the help of high-frequency piston pumps, not only can a small amount of gas be delivered and extracted from the airway to achieve lung-protective ventilation, but it also avoids barotrauma and enhances oxygenation. Chen Yuhang et al. [13] found that mechanical ventilation can provide respiratory support for children, but increased airway pressure and tidal volume can damage lung tissue, impair alveolar structure, and as mechanical ventilation duration increases, natural respiratory defense mechanisms are affected, leading to more severe inflammatory responses. However, high-frequency oscillatory ventilation can assist pulmonary gas exchange, reduce airway pressure and oxygen requirements, promote effective oxygenation and carbon dioxide removal, and under its influence, can facilitate alveolar re-expansion, improve gas distribution in lung tissue, and prevent average airway descent, which can lead to alveolar collapse.

4.3 Hormone therapy

Wang Lirong's [14] study found that the use of glucocorticoids before birth can thin the interstitial tissue in the fetal lungs, promoting faster lung maturation and thus improving lung function. This can better protect newborns from pulmonary diseases and help reduce neonatal mortality. Additionally, administering systemic glucocorticoids to newborns after birth can decrease pulmonary capillary permeability, aiding in the improvement of bronchial and pulmonary edema in affected infants. It also effectively suppresses pulmonary inflammatory responses, enhances β -receptor agonist responses, and promotes PS synthesis, which helps accelerate the recovery of bronchopulmonary dysplasia lung function and allows for earlier weaning from mechanical ventilation.

4.4 Mesenchymal stem cells

Mesenchymal stem cell (MSCs) therapy is a new treatment for bronchopulmonary dysplasia in recent years, providing a new therapeutic approach for patients. Dong Mengyuan et al. [15] found that mesenchymal stem cells have a good preventive effect in the treatment of bronchopulmonary dysplasia. By utilizing matrix proteins, exosome paracrine signaling, and cell derivatives, they can effectively improve bronchopulmonary dysplasia. Derivatives can achieve better therapeutic effects, effectively improving alveolar simplification, fibrosis, and correcting pulmonary vascular remodeling, which helps promote the rapid recovery of lung function and prevent pulmonary hypertension.

5 Conclusion

In recent years, there have been significant advancements in neonatal care and perinatal medicine. However, the incidence, disability rate, and mortality rate of bronchopulmonary dysplasia remain high. There is currently no specific treatment or preventive measure for bronchopulmonary dysplasia. Although there are various treatment options available, they still face some controversy. In the future, further research is needed to implement comprehensive treatments based on the individual circumstances of patients, thereby reducing the incidence of complications associated with bronchopulmonary dysplasia and achieving better therapeutic outcomes.

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